# DISSERTATION

# ON

# "A STUDY OF CLINICAL AND ELECTROPHYSIOLOGICAL PROFILE OF GUILLAIN-BARRÉ SYNDROME"

*Submitted in partial fulfilment of*

*requirements for the degree of*

**D.M. NEUROLOGY (BRANCH – I)**

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# CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF CLINICAL AND ELECTROPHYSIOLOGICAL PROFILE OF GUILLAIN-BARRÉ SYNDROME"** submitted by **Dr. R.ARUNAGIRI** appearing for **D.M. Neurology** Degree (Branch - I) examination in **August 2011** is a bonafide record of work dosne by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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# DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF CLINICAL AND ELECTROPHYSIOLOGICAL PROFILE OF GUILLAIN-BARRÉ SYNDROME"** is done by me at Government Rajaji hospital,Madurai during 2008-2011 under the guidance and supervision of **Prof. Dr. N.MUTHUVEERAN,M.D,D.M**

The dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of **D.M., degree in Neurology.**

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## LIST OF ABBREVATIONS

**GBS -** Guillain Barre Syndrome

**AIDP -** Acute inflammatory demyelinating polyneuropathy

**CSF -** Cerebro Spinal Fluid

**NINCDS**-National Institute of Neurological and Communicative Disorders and Stroke

**SNAP -** Sensory Nerve Action Potential

**CMAP-** Compound Muscle Action Potential

**IG -** Immunoglobulin

# INTRODUCTION

Guillain Barre Syndrome (GBS) or Acute Polyradiculoneuritis is an acute, diffuse post infective disorder of the nervous system involving the spinal roots, the peripheral nerves and occasionally the cranial nerves. The aetiology is thought to be widespread demyelination of the spinal roots and the peripheral nerves due to a cross reaction between myelin and unconfirmed agents like viruses. GBS is a syndrome of acute areflexic motor paralysis. The disorder is heterogeneous and diverse in its antecedent events, clinical presentations and natural course, such that making the diagnosis is a challenge for most neurologists. Although GBS often has a benign prognosis, 7% of patients die and further 16% suffer residual disability. The modalities of treatment of GBS are physiotherapy, supportive treatment, ventilator management, plasma exchange and of late intravenous immunoglobulins.

This thesis was undertaken to study the clinical and epidemiological profile as well as electrophysiological features of Guillain Barre Syndrome and to know the incidence of various variants in the studied population.

# AIM OF THE STUDY

- To study the epidemiological features of GBS
- To analyze the clinical profile of GBS
- To evaluate electrophysiological features of GBS
- To know about various G B variants in studied population

# REVIEW OF LITERATURE

Guillain-Barré syndrome (GBS) is an acute, frequently severe, and fulminant polyradiculoneuropathy that is autoimmune in nature. It is the most common cause of acute or sub acute generalized paralysis in practice. (During certain past epochs it was exceeded in frequency by polio.)[1]

**Historical Background**

As with many other neurological syndromes, the understanding of G B syndrome has been shaped by the historic sequence of its description and contemporary developments in medicine

Octave Landry is credited with the earliest description of what is now recognized as GBS. In 1859 he described a condition called **'acute ascending paralysis'.**[7]

In 1916, G. Guillain, Barre, and Strohl - French army neurologists, reported on two soldiers who developed an acute paralysis associated with the loss of muscle stretch reflexes. They also described an elevation of CSF

protein with a normal cell count (albuminocytological dissociation). Andre Strohl was responsible for the electrophysiological aspects.[1]

Landry's paralysis and Guillain Barre Syndrome were thought to be different entities (with a benign prognosis for GBS and an ominous prognosis for Landry's paralysis) till Haymaker and Kernohan in 1949,[8] exhaustively reviewed this debate and entitled their paper Landry-Guillain-Barre syndrome implying that the two conditions are identical. Miller Fisher in 1956 observed a variant associated with ophthalmoplegia,ataxia, and areflexia.[9]

**EPIDEMIOLOGY**: GBS occurs year-round at a rate of about 0.6-1.9 cases/100,000 population. GBS occurs in all parts of the world and in all seasons, affecting children and adults of all ages and both sexes. Males are at 1.5-fold higher risk for GBS than females, and in western countries adults are more frequently affected than children. Slight increased frequency observed in Caucasians.[1, 2, 5]

## Historical Background

| Year | Developments |
|---|---|
| 1834-1837 | Earliest description of an afebrile generalized paralysis by Wardrop and Ollivier |
| 1859 | Landry's report of an acute, ascending, predominantly motor paralysis with respiratory failure leading to death |
| 1892 | Osler's description of "febrile polyneuritis"; |
| 1916 | The eponym “GBS” derives it’s name from the description by George Guillaine Barre and Strohl who performed the electrophysiological studies & CSF studies |
| 1949 | The first comprehensive account of the pathology of GBS by Haymaker and Kernohan |
| 1969 | Asbury and colleagues established that the essential lesion was perivascular mononuclear inflammatory infiltration of the roots and nerves |
| 1975 | Swine flu vaccination programme spurs interest G B syndrome |
| 1978 | Use of plasmaphresis in treatment reported by Brettle and coworker |
| 1990 | NINCDS criteria for G B syndrome |

**ETIOLOGY: A** mild respiratory or gastrointestinal infection or immunization precedes the neuropathic symptoms by 1 to 3 weeks in approximately 60 percent of cases. Typical is a nondescript upper respiratory infection but almost every known febrile infection and immunization has at one time or another been reported to precede GBS . Respiratory infections are most frequently reported, followed by gastrointestinal infections

In recent years, it has been appreciated from serologic studies that the enteric organism *Campylobacter jejuni* is the most frequent identifiable antecedent infection reported in up to 32% of cases. Clinical enteritis may be absent in 30% of *C.jejuni* associated GBS. In these cases there is only serologic evidence of the prior bacterial infection. Patients who develop GBS following an antecedent *C jejuni* infection often have a more severe course, with rapid progression , elevated anti-GM1 antibodies, and a prolonged, incomplete recovery. A strong clinical association has been noted between *C jejuni* infections and the pure motor and axonal forms of GBS.[18]

Other common antecedent events or associated illnesses include

viral exanthems in children and numerous other viral illnesses in adults and children,[6] particularly the large viruses of the herpes family (cytomegalovirus [CMV], Epstein-Barr virus [EBV].[19] Cytomegalovirus (CMV) is the second most common antecedent infection with serologic evidence in up to 15% of cases a. CMV induced GBS tends to occur in younger patients and is often severe with respiratory failure, marked sensory and cranial nerve dysfunction, and elevated antibodies against ganglioside GM2. Epstein Barr (EBV) infection may precede GBS in about 10% of cases; preceding clinical signs include mononucleosis, hepatitis, or pharyngitis.GBS may occur with HIV seroconversion.[1, 2, 5]

Other significant, although less frequently identified, infectious agents in GBS patients include Mycoplasma pneumoniae, Lyme disease, Haemophilus influenzae, para-influenza virus type 1, influenza A virus, influenza B virus, adenovirus, and herpes simplex virus have been demonstrated in patients with GBS .Hodgkin's disease, lung cancer, thyroid disease, SLE, paraproteinemia, and sarcoidosis,surgery, trauma, and in the post-partum period.

The administration of older-type rabies vaccine, prepared in nervous system tissue, is implicated as a trigger of GBS in developing countries where it is still used and A/New Jersey (swine) influenza

vaccine was associated with a slight increase in the incidence of GBS. Cases are seen in temporal relationship to almost all other vaccinations, but the association in these instances seems idiosyncratic and infrequent.

## PATHOPHYSIOLOGY

GBS is a post infectious, immune-mediated disease. It is likely that both cellular and humoral immune mechanisms contribute to tissue damage in AIDP. T cell activation is suggested by the finding that elevated levels of cytokines and cytokine receptors are present in serum [interleukin (IL) 2, soluble IL-2 receptor] and in cerebrospinal fluid. Most patients report an infectious illness in the weeks prior to the onset of GBS. Many of the identified infectious agents are thought to induce antibody production against specific gangliosides and glycolipids, such as GM1 and GD1b, [10] distributed throughout the myelin in the peripheral nervous system. The pathophysiologic mechanism of an antecedent illness and of GBS can be typified by campylobacter jejuni infections. The virulence of C.jejuni is thought to be based on the presence of specific antigens in its capsule that are shared with nerves. Immune responses directed against the capsular components produce antibodies that cross-react with myelin to cause demyelination.[11] Ganglioside GM1 appears to cross-react with *C jejuni* lipopolysaccharide antigens, resulting

in the immunologic damage to the peripheral nervous system. This process has been termed molecular mimicry.[12]

Classic pathophysiological studies of AIDP have demonstrated endoneurial perivascular mononuclear cell infiltration followed by macrophage – mediated ,multifocal stripping of myelin. This phenomenon results in defects in the propagation of electrical nerve impulses, with eventual; conduction block and flaccid paralysis.[14-16]In some patients with severe disease, a secondary consequence of the severe inflammation is axonal disruption and loss. A subgroup of patients may have a primary immune attack directly against nerve axons ,resulting in a similar clinical presentation.[17,18] The peripheral nerves may be affected at all levels from the roots to distal intramuscular motor nerve findings, although the brunt of the lesions frequently falls on the ventral roots ,proximal spinal nerves and cranial nerves[20]

**Principal Anti-Glycolipid Antibodies Implicated in Acute Immune Neuropathies[11]**

| Clinical Presentation | Antibody Target | Usual Isotype |
|---|---|---|
| Acute inflammatory demyelinating polyneuropathy (AIDP) | No clear patterns<br>GM1 most common | IgG (polyclonal) |
| Acute motor axonal neuropathy (AMAN) | GD1a, GM1, GM1b, GalNAc–GD1a (<50% for any) | IgG (polyclonal) |
| Miller Fisher syndrome (MFS) | GQ1b (>90%) | IgG (polyclonal) |
| Acute pharyngeal cervicobrachial neuropathy APCBN) | GT1a | IgG (polyclonal) |

***Source:*** Modified from HJ Willison, N Yuki: Brain 125:2591, 2002.

## CLINICAL MANIFESTATIONS

GBS manifests as rapidly evolving areflexic motor paralysis with or without sensory disturbance. The usual pattern is an ascending paralysis that may be first noticed as rubbery legs. Weakness typically evolves over hours to a few days and is frequently accompanied by tingling dysesthesias in the extremities. The legs are usually more affected than the arms, the trunk, intercostal, neck, and cranial muscles may be affected later. Facial diparesis is present in 50% of affected

individuals[21, 22]. The lower cranial nerves are also frequently involved, causing bulbar weakness with difficulty handling secretions and maintaining an airway; the diagnosis in these patients may initially be mistaken for brainstem ischemia[23]. Ophthalmoparesis may be observed in up to 25% of patients with GBS. Limitation of eye movement most commonly results from a symmetric palsy associated with cranial nerve VI. Ptosis from oculomotor nerve palsy also is often associated with limited eye movements Weakness progresses in approximately 5 percent of patients to total motor paralysis with respiratory failure within a few days[24] .

Pain in the neck, shoulder, back, or diffusely over the spine is also common in the early stages of GBS, occurring in ~50% of patients. These symptoms precede weakness and may be mistaken for lumbar disc disease, back strain, and orthopedic diseases. Deep tendon reflexes attenuate or disappear within the first few days of onset. Cutaneous sensory deficits (e.g., loss of pain and temperature sensation) are usually relatively mild, but functions subserved by large sensory fibers, such as deep tendon reflexes and proprioception, are more severely affected[25, 26]. Bladder dysfunction may occur in severe cases but is usually transient. If bladder dysfunction is a prominent feature and comes early in the course,

diagnostic possibilities other than GBS should be considered, particularly spinal cord disease. Once clinical worsening stops and the patient reaches a plateau (almost always within 4 weeks of onset), further progression is unlikely.

Autonomic involvement is common and may occur even in patients whose GBS is otherwise mild. Autonomic dysfunction of various degrees has been reported in 65% of patients admitted to the hospital. Its manifestations may be related to either increased or decreased sympathetic activity[27]. Signs of decreased sympathetic {orthostatic hypotension, anhidrosis) or decreased parasympathetic {urinary retention, gastrointestinal atony, or iridoplegia) function may be seen. Signs of excessive sympathetic activity include episodic or sustained hypertension, sinus tachycardia, tachyarrhythmias, episodic diaphoresis, and acral vasoconstriction. Excessive vagal activity accounts for sudden episodes of bradycardia, heart block, and asystole. These features require close monitoring and management and can be fatal. Urinary retention occurs in approximately 15 percent of patients soon after the onset of weakness, but catheterization is seldom required for more than a few days.

Most patients require hospitalization, and almost 30% require ventilatory assistance at some time during the illness. Patients with

weakness of neck muscles, tongue and palate often have concomitant diaphragmatic and respiratory muscle involvement. Patients requiring ventilator support have less favorable prognosis for neurologic recovery, have longer hospitalizations, and higher mortality. Fever and constitutional symptoms are absent at the onset and, if present, cast doubt on the diagnosis. Recovery usually begins 2-4 weeks after the progression ceases

**GBS VARIANTS:**

Several subtypes of GBS are recognized, as determined primarily by electro diagnostic and pathologic distinctions [3,4,29].

**ACUTE MOTOR AND SENSORY AXONAL NEUROPATHY (AMSAN):**

Initially described by Feasby as axonal GBS. Characterized by acute quadriparesis, areflexia, distal sensory loss, and respiratory insufficiency. CSF examination shows increased protein. Electro diagnosis shows loss of motor and sensory potentials with diffuse active denervation. AMSAN leads to severe with quadriplegia, respiratory insufficiency and delayed, incomplete recovery[30]

**ACUTE MOTOR AXONAL NEUROPATHY (AMAN) :**

Originally described in patients from northern China,

particularly children. It is characterized by acute/sub acute onset of relatively symmetric limb weakness, diffuse areflexia, facial and oropharyngeal muscle weakness, and respiratory insufficiency. Clinically presents as pure motor deficits[30]. Often associated with gastric enteritis due to *C. jejuni* with elevated anti-GM1 and anti-GD1a antibodies to *C. jejuni*. Electro diagnostic studies show evidence of motor axon loss sparing sensory Fibers without evidence of demyelination. Needle EMG shows diffuse denervation. CSF examination shows increased protein levels.

**MILLER-FISHER VARIANT:**

The Miller-Fisher syndrome, a common variant of GBS, is observed in about 5% of all GBS cases It is characterized by Classic triad of ophthalmoplegia, ataxia, and areflexia described by C. Miller Fisher in 1956. Diplopia is the usual initial symptom, followed by limb or gait ataxia and areflexia. Abducens nerve palsy is the earliest lesion which may progress to complete ophthalmoplegia and ptosis, but pupillary reflex is spared. Ataxia is primarily noted during gait and in the trunk, with lesser involvement of the limbs. Motor strength is characteristically spared.Occasionally there may be mild sensory symptoms, swallowing difficulties, or proximal limb weakness in up to 1/3 or 1/2 of cases. The

usual course is one of gradual and complete recovery over weeks or months. Although CSF protein is mildly elevated, it is less so than in typical GBS. Electro diagnosis shows loss of sensory potentials, with milder axonal degeneration. It clinically resembles brainstem inflammatory or ischemic disease. A close association exists between antiganglioside antibodies and the Fisher variant. Anti-GQ1b antibodies, triggered by certain *C jejuni* strains, have a relatively high specificity and sensitivity for the disease.[6]Dense concentrations of GQ1b ganglioside are found in the oculomotor, trochlear, and abducens nerves, which may explain the relationship between anti-GQ1b antibodies and ophthalmoplegia

**PURE MOTOR VARIANTS:**

Acute, progressive, symmetric limb weakness, predominantly distal limb weakness,areflexia, without sensory loss. Investigation shows elevated anti-GM1 titers due to preceding *C. jejuni* infection. Course and recovery is similar to typical GBS. CSF protein is elevated. Electro diagnosis shows marked axonal degeneration with some accompanying demyelinating features. Differential diagnosis includes poliomyelitis, porphyria, acute fulminant, myasthenia gravis, tick paralysis.

## PURE SENSORY VARIANTS:

A pure sensory variant of GBS has been described in the medical literature, typified by a rapid onset of large fiber sensory loss with resultant sensory ataxia and areflexia in a symmetric and widespread pattern. Sensory dysfunction may involve the face and torso in severe cases. Lumbar puncture studies show albuminocytologic dissociation in the cerebrospinal fluid (CSF), and electromyography (EMG) results show characteristic signs of a demyelinating process in the peripheral nerves. Prognosis is generally good, but immunotherapies, such as plasma exchange and the administration of intravenous immunoglobulin (IVIGs), can be tried in patients with severe disease or slow recovery. Differential diagnosis includes cervical myelopathy, malignant and nonmalignant sensory neuronopathy, Sjogren's syndrome, ciguatera poisoning.

## PURE DYSAUTONOMIA VARIANT:

Acute pandysautonomia without significant motor or sensory involvement is a rare presentation of GBS, **first** described by Young and coworkers, as a variant. Dysfunction of the sympathetic and parasympathetic systems results in severe postural hypotension, bowel

and bladder retention, anhidrosis, decreased salivation and lacrimation, and pupillary abnormalities. About half of the patients have autoantibodies to ganglionic acetylcholine receptors which may play a pathogenetic role by blocking cholinergic transmission in autonomic ganglia. Routine Electro diagnosis studies are normal; autonomic testing such as heart rate variability, tilt-table testing, sympathetic skin responses, and sweat testing (QSART) may be abnormal. The condition characteristically progresses and then plateaus after few weeks. 50% of patients may recover slowly after several months.

**PHARYNGEAL-CERVICAL-BRACHIAL VARIANT:**

The pharyngeal-cervical-brachial variant is distinguished by isolated facial, oropharyngeal, cervical and upper limb weakness without lower limb involvement. (Ropper, 1986a). Ptosis, often with ophthalmoplegia, may be present. High titers to GT1a antibodies may present in few cases. CSF protein is elevated. Electro diagnostic studies may be normal, or show demyelinating changes in upper limbs. Recovery is delayed and, at times, incomplete. The differential diagnosis includes myasthenia gravis, diphtheria, and botulism and a lesion affecting the central portion of the cervical spinal cord and lower brainstem

**PARAPARETIC VARIANT:**

It is a type of regional variant with isolated leg weakness and areflexia. Upper limbs, cranial nerves and sphincters are spared. Radicular pain commonly occurs in these patients. CSF shows elevated protein levels. Electro diagnosis shows demyelinating changes in lower limb nerves. MRI of spinal cord and lumbar roots is necessary to exclude lesion of distal cord and cauda equina

**OTHER LESS COMMON VARIANTS:**

Portions of the clinical picture frequently appear in isolated or abortive form and are a source of diagnostic confusion

i. Acral paresthesias with diminished reflexes in either arms or legs.
ii. Facial diplegia or abducens palsies with distal paresthesias
iii. Isolated post infectious ophthalmoplegia.
iv. Bilateral foot-drop with upper limb paresthesias.
v. Acute ataxia without ophthalmoplegia.
vi. Pure ophthalmoplegic form- associated with a specific anti-$GQ_1b$ antibody

vii. GBS with severe bulbar and facial paralysis- associated with antecedent cytomegalovirus infection and anti-GM2 antibodies[75]

viii. Acute polyneuritis cranialis not involving the first or second cranial nerves, may be a variant of GBS, when there is a monophasic illness with acute onset, raised CSF protein and recovery and no other cause is found

Less difficulty attends the correct diagnosis of GBS if paresthesias in the acral extremities, progressive reduction or loss of reflexes and relative symmetry of weakness appear after several days. The laboratory tests, particularly nerve conduction studies that affirm the diagnosis of typical GBS, give similar but generally milder abnormalities if they are carefully sought in all these variant forms. In a few patients, the weakness continues to evolve for 3 to 4 weeks or longer. From this group, a chronic form of demyelinative neuropathy (CIDP) may emerge and an intermediate group that progresses for 4 to 8 weeks and then improves can be identified

**DIAGNOSIS:**

GBS is a descriptive entity. The diagnosis is made by recognizing the pattern of rapidly evolving paralysis with areflexia, absence of fever or other systemic symptoms, and characteristic antecedent events. Other disorders that may enter into the differential diagnosis include acute myelopathies (especially with prolonged back pain and sphincter disturbances); botulism (pupillary reactivity lost early); diphtheria (early oropharyngeal disturbances); Lyme polyradiculitis and other tick-borne paralyses; porphyria (abdominal pain, seizures, psychosis); vasculitic neuropathy (elevated erythrocyte sedimentation rate); poliomyelitis (fever and meningismus common); CMV polyradiculitis (in immunocompromised patients); critical illness neuropathy; neuromuscular disorders such as myasthenia gravis; poisonings with organophosphates, thallium, or arsenic; tick paralysis; paralytic shellfish poisoning; or severe hypophosphatemia (rare). Laboratory tests are helpful primarily to exclude mimics of GBS.

**LABORATORY FINDINGS:**

The most important laboratory aids are the electro diagnostic studies and CSF examination.

The CSF is often normal when symptoms have been present for 48 h and by the end of the first week the level of protein is usually elevated

with levels up to 100–1000 mg/dL, under normal pressure without accompanying pleocytosis. CSF protein level rises, reaching a peak in 4 to 6 weeks and persisting at a variably elevated level for many weeks. The increase in CSF protein is probably a reflection of widespread inflammatory disease of the nerve roots, but high values have had no clinical or prognostic significance .In a few patients (fewer than 10 percent), the CSF protein values remain normal throughout the illness. In 10 percent of patients 10 to 50 cells per cubic millimeter, predominantly lymphocytes, may be found. Persistent pleocytosis suggests an alternative or additional process producing aseptic meningitis such as neoplastic infiltration, HIV, or Lyme infection. Both tau and 14-3-3 protein levels are reported to be elevated early (during the first few days of symptoms) in some cases of GBS. Tau increases in CSF may reflect axonal damage and predict a residual deficit.

Abnormalities of nerve conduction are early and dependable diagnostic indicators of GBS[4]. They are diagnostic in 95% cases. Absent H-reflexes, delayed or absent or impersistent F waves, and low amplitude or absent SNAPs in the upper extremity combined with normal sural SNAPs are changes supportive of the diagnosis in the first week of illness. Typically, there is multifocal demyelination affecting proximal

and distal nerve segments. Prolonged or absent F waves may be initial sole abnormality in about 30-50% of cases .Evidence of conduction block occurs in about 1/3 of cases; conduction slowing and temporal dispersion reflect demyelination. Prolonged distal latencies (reflecting distal conduction block) and absent F waves or prolonged F latency (indicating involvement of proximal parts of nerves and roots) are important diagnostic findings, all reflecting focal areas of demyelination.

Most important predictor of recovery is the degree of axonal degeneration, best reflected by the amplitude of the compound muscle action potential. Motor potentials with amplitudes less than 20% normal suggest a prolonged, and often incomplete recovery.

Needle EMG initially shows decreased motor unit recruitment. Subsequently with axonal degeneration fibrillation potentials appear 2—4 weeks after onset.

Electro diagnostic studies performed in the patients enrolled in the North American GBS Study found abnormalities of distal motor latencies and F-wave latencies in approximately one half of patients studied within 30 days of onset. Partial motor conduction block (30%), slowing of motor conduction velocity (24%), and reduced distal CMAP amplitudes (20%) were less frequent.[3, 4]

Lumbosacral spinal MRI may demonstrate gadolinium

enhancement of lumbar roots in few cases.

**DIAGNOSTIC CRITERIA FOR GBS (**Asbury And Cornblath*)* [35, 36]

I. ***Features required for diagnosis***

a. Progressive motor weakness of more than one limb.

b. Areflexia

II. ***Features strongly supportive of the diagnosis***

a. Clinical features:

i. Progression within four weeks

ii. Relative symmetry of symptoms

iii. Mild sensory symptoms or signs

iv. Cranial nerve involvement

v. Recovery within four weeks of progression cessation

vi. Autonomic dysfunction

vii. Absence of fever at onset

b. CSF picture:

i. Raised CSF protein after 1 week of symptoms

ii. Cell counts of less than or equal to 10 mononuclear leucocytes per cmm of CSF

c. Electro diagnostic studies:

i. Electro diagnostic features strongly supportive of the diagnosis (nerve conduction slowing or block)

III. ***Features casting doubt on the diagnosis***

i. Pronounced persistent asymmetry of weakness
ii. Persistent bladder or bowel dysfunction
iii. Bladder or bowel dysfunction at onset
iv. More than 50 mononuclear leucocytes/$^{3}$ mm
v. Presence of polymorphonuclear leucocytes in CSF
vi. Sharp sensory level

IV. ***Features that rule out the diagnosis***

i. Current history of hexacarbon misuse
ii. Abnormal porphyrin metabolism
iii. Recent diphtheritic infection
iv. Features clinically consistent with lead neuropathy
v. Purely sensory syndrome
vi. Definite diagnosis of poliomyelitis, botulism, hysterical paralysis, or toxic neuropathy

## ELECTRODIAGNOSTIC CRITERIA FOR GBS [3,4,77]

### Electrophysiological classification of Guillain-Barre syndrome

At least 3 sensory nerves and 3 motor nerves with multi-site stimulation F waves, and bilateral tibial H reflexes need to be evaluated.

**AIDP**

At least 1 of the following in each of at least 2 nerves, or at least 2 of the following in 1 nerve if all others inexcitable and distal compound muscle action potential (dCMAP) >10% lower limit of normal (LLN):

- Motor conduction velocity <90% LLN (85% if dCMAP <50% LLN)
- Distal motor latency >110% upper limit of normal (ULN) (>120% if dCMAP <100% LLN)
- pCMAP/dCMAP ratio <0.5 and dCMAP >20% LLN
- F-response latency >120% ULN.

**AMSAN**

- None of the features of AIDP except 1 demyelinating feature allowed in 1 nerve if dCMAP <10% LLN
- Sensory action potential amplitudes less than LLN.

**AMAN**

- None of the features of AIDP except 1 demyelinating feature allowed in 1 nerve if dCMAP <10% LLN
- Sensory action potential amplitudes normal.

**Inexcitable**

- dCMAP absent in all nerves or present in only 1 nerve with dCMAP <10%.

**TREATMENT:**

All patients with suspected GBS should be hospitalized for vigilant monitoring due to the high risk of respiratory failure and need for intubation and mechanical ventilation. Baseline spirometry, including FVC and oximetry, should be obtained. The cornerstone of treatment is that of meticulous general medical support.

Very mild cases with only distal paresthesia and mild limb weakness may not need treatment, but it is advisable to wait approximately two weeks before concluding that there will be no further progression. Patients with vital capacities that are rapidly declining or below 18ml/kg or those with cardiovascular dysautonomia are appropriate candidates for observation in an ICU.

The most important advances in treatment of GBS have been positive pressure ventilation and intensive respiratory and medical management in the ICU, both introduced during the European Poliomyelitis Epidemic of the 1950s. These have allowed patients with complications of immobilization and respiratory failure to survive and recover from paralysis.

***The criteria advocated for intuabation, weaning and extubation in GBS are:***

**A. Intubation:**

**i.** Mechanical ventilatory failure with vital capacity (VC) of 12-15 ml/kg.Falling VC over 4-6 hours or clinical signs of fatigue (brow sweating, tachycardia, and hyperpnoea) may prompt intubation at 15ml/kg.

**ii.** PaO2 less than 70mmHg on inspired air.

**iii.** Severe oropharyngeal paresis with difficulty in clearing secretions or repeated coughing and aspiration after swallowing.

**B. Weaning from ventilator:**

i. When VC exceeds 8-10 ml/kg with adequate oxygenation maintained with 35- 40% inspired oxygen.

**ii.** Patient can voluntarily double resting minute volume.

**C. Extubation:**

1. When continuous positive airway pressure of 5-7 cm of water was tolerated without clinical signs of fatigue for 12-24 hours.
2. Arterial Pao2 greater than 9O mmHg on room air.
3. Adequate alveolar ventilation.
4. Improvement in bulbar paresis.

Although many patients of GBS have clinical signs of fatigue of respiratory muscles, only 10% - 30% patients eventually require mechanical ventilation. Indications for intubation include an FVC dropping below 12ml/kg or in a normalized adult, FVC falling below 1 liter patients who are subjectively dyspneic or appear to be struggling to breathe should be intubated, even if their FVC is above these levels. In general one should err on the side of early intubation than late. A simple bedside estimate of FVC can be made by having the patient count out loud. If the patient can take a maximal inspiration and then can count up to 25, the FVC is probably about 2 liters. A patient who can count up to 10 probably has about 1 liter

FVC.

In patients with respiratory failure, the average duration of machine assisted respiration has been 50 days. In a study form India, 30% of the patients studied required mechanical ventilation. Weakness of facial,

truncal, neck, bulbar and proximal weakness of upper limb and autonomic disturbances were predictors for need for subsequent mechanical ventilation.

The prevention of nosocomial infections is another central feature of treatment, since 25% of patients acquire pneumonias and 30% acquire urinary infections. Prophylaxis for pulmonary embolism, adequate nutrition, and psychological care are the other major areas of concern in severe cases. As many as 30% of patients with GBS, experience significant pain early in their presentation. In adults pain tends to be in the paraspinal region. In children, pain may be more pronounced in the limbs. Conventional analgesics may be useful, in addition to those commonly used for treating neurogenic pain (such as tricyclic antidepressent medications). Alternatively, a brief course of high-dose corticosteroids can lead to marked improvement in pain control.

**IMMUNOTHERAPY OF GBS:**

**Corticosteroids:**

For 50 years corticosteroids were the main stay of treatment for acute GBS on the basis of anecdotal experience, a few uncontrolled studies and the appeal of their anti-inflammatory effect[40].

Two randomized controlled trails, one using conventional doses of prednisolone for two weeks[41] and the other, use high dose (500 mg)

intravenous methylprednisolone [42] daily for 5 days have found no benefit. Steroids are thus no longer considered useful for **GBS.**

**Plasma Exchange:**

Evidence of the presence of antibodies[43] or demyelinating serum factors has provided a rationale for use of plasma exchange in therapy of GBS.[13] In 1978, Brettel et al[44] first reported the benefits of plasma exchange in the treatment of GBS. Since then, several trials have been confirmatory. Three large trials - The North American GBS Study Group[45], the French[46] and the Swedish[13] trials have established the benefit of plasma exchange. The conclusions derived from these three trials were: Plasma exchange is beneficial in acute GBS; it favorably modifies poor prognostic factors; maximum benefit is obtained when it is instituted early (within two weeks of illness) and not much benefit is expected after three weeks.

In the usual regimen of plasma exchange, [21] a total of 200-250ml of plasma per kg body weight is removed in 4 - 6 treatments on alternate days. The time required to complete a series of exchanges is 8 - 14 days. Plasma as replacement fluid is not recommended [46] and now a days albumin or saline is used. The type of replacement fluid (albumin / saline) used in plasma exchange has not been found to influence the outcome

Patients undergoing plasma exchange on continuous flow machine

have a better outcome than on intermittent flow machines.[33] Two continuous flow techniques can be used: Ultra filtration and centrifugation. In a recent study, no difference was found in terms of outcome between these two techniques.[47]

Indications for starting plasma exchange in patients of GBS have been advocated by Mckhann and Griffin.[48] These are: inability to walk unaided; rapid and significant reduction in serial vital capacity and onset of bulbar paralysis. The benefit of plasma exchange is diminished if treatment is begun after two weeks of illness but some patients still seem to benefit if their condition continued to worsen during third week. After an initial good response with plasma exchange, some patients may show deterioration necessitating further series of plasma exchange[33, 34].

Complications of plasma exchange[49] may be related to the procedure (hypotension, volume overload), vascular access (venous thrombosis, pneumothorax with subclavian catheter), anti coagulation (bleeding tendencies with heparin, depletion of coagulation factors), replacement fluids (anaphylaxis with plasma usage) [39]. Plasma exchange also has certain practical limitations like: availability of technical set up and trained personnel to carry out the procedure; greater risk for patients with cardiovascular complications or marked dysautonomia (associated with GBS) and small but real risk of transfer of infections (hepatitis and

HIV) when plasma is used as replacement fluid[38].

**Modified Plasma Exchange:**

A study from India[50] showed that small volume plasma exchange (10 - 15ml/kg of plasma exchanged on consecutive days) during first two weeks of the illness showed results comparable to that of conventional plasma exchange (200 -250 ml/kg). A similar study report from Sri Lanka[61] has supported this fact. All this was achieved at a lower cost and fewer complications which is important for widespread application in developing countries. The replacement fluid used in these studies was fresh frozen plasma.

**Immunoglobulins**

High dose intravenous immunoglobulin was first reported to be beneficial in GBS by Kleywey et al[52] in 1988.Intravenous immunoglobulin has been found to be useful in treatment of many diseases with an autoimmune basis[37]. The proposed mechanisms[53] for their action are:

a. Passive transfer of neutralizing anti-idiotypic antibodies against auto antibodies.
b. Intravenous immunoglobulin alters the structure and dynamics of the idiotypic network in the auto immune patient to regain physiological control of auto immunity.

During the first week of illness, the preferred treatment for patients with GBS who have severe disease and require assistance to walk is either plasmapheresis or intravenous immunoglobulin. A controlled, randomized trial[47] comparing intravenous immunoglobulin treatment with plasma exchange concluded that intravenous immunoglobulin was also effective as first line treatment. In this study, 52.7% of 74 patients receiving immunoglobulin compared with 34% of 73 patients undergoing plasma exchange had functional improvement of one grade or more after 4 weeks. Although immunoglobulin was clearly more efficacious, this findings was challenged because response to plasma exchange in this study[47] was inferior to that in earlier studies.[45] This prompted a new multinational, multicentre trial[54] that compared efficacy of immunoglobulin alone, plasmapheresis alone and plasmapheresis followed by immunoglobulin. After 4 weeks of treatment and 48 weeks of follow up, no statistically significant difference was seen between the treatments. Consequently the question of which therapy is preferable for using first considering their similar efficacy and cost is a matter of convenience and practicality.[55]

The indications for immunoglobulin in GBS have not been clearly laid down yet. In the Dutch study trial, [47] inclusion criteria included acute GBS, inability to walk 10m independently and presentation within 2

weeks of disease onset. If the illness continues to worsen in the first 15 days despite a full course of plasma exchange, a trial of immunoglobulin therapy [55] is warranted. The dose recommended is 0.4g/kg/day for 5 days. Just like plasma exchange, the use of immunoglobulin has also been associated with treatment related fluctuations.

Adverse reactions[55] to immunoglobulin therapy are usually minor and occur in about 10% of patients. Side effects include fever, myalgia, headache, meningism, and eczema on hands. There is also an increased risk of thromboembolic events, as therapy with immunoglobulin increases serum viscosity. Anaphylaxis is very rare but potentially fatal. Immunoglobulin should be used with extreme caution in patients with renal failure which it may exacerbate.

**REHABILITATION:**

Rehabilitation requires an organized program with definite end points. There are no systematic studies of physiotherapy in GBS. But early goals include prevention of decubitus ulcers, tendon shortening, joint malalignment, peroneal nerve compression palsies and facilitation of pulmonary toilet. Psychological problems in form of depression, mental fatigue, impotence and those due to residual weakness should also be tackled.

**OUTCOME:**

Although GBS is often thought to have a benign prognosis, 7% of patients die and a further 16% suffer significant residual disability.[66] Speed of recovery varies and may take 6-8 months; no improvement is expected after 2 years.[57] Remyelination is usually complete but recovery is poor when there has been Wallerian axonal degeneration [58] after which the axons regrow at 1mm per day so many do not reach their target muscles for several years if at all.

The death rate has varied from 1.3% in different series with a mean of 6%, probably depending more on the quality of intensive care than specific treatments. Half of the deaths [58] are within first month - one third from cardiovascular autonomic complications such as asystole; a quarter from pneumonia or respiratory failure and the rest from pulmonary embolism, infection, infarction, renal failure and unrelated cause.

With the currently available modalities of treatment, it is estimated that about 15% of GBS patients completely recover without any deficit and another 65% have persistent minor problems that generally do not impair conduct of everyday life.[21] The disease in itself does not result in chronic fatigue but mild depression indicated by persistent mental fatigue is common. A few men have residual impotence. Three percent of patients may have one or more recurrence[57] of the disease.

**COMPLICATIONS:**

The common complications of GBS include respiratory failure, atelectasis, aspiration, pneumonia, bladder dysfunction, pain, depression, phlebitis, pulmonary embolus and syndrome of inappropriate antidiuretic hormone secretion (SIADH) exacerbated by positive pressure ventilation. Pseudo tumor cerebri, papilloedema alone and unexplained seizures have been reported.

Further complications from immobility and bed rest include decubitus ulcers secondary compression neuropathy such as ulnar neuropathy and peroneal neuropathy, and the psychiatric sequelae associated with a prolonged, immobilizing stay in the ICU.

**PROGNOSIS:**

Although the majority of patients with GBS make an acceptable functional recovery, a proportion succumb to the acute illness while others retain significant residual disability. It is thus important to be able to select only patients with a poor prognosis for treatment, which often involves discomfort, expense and risk of complications. No large-scale prospective study has ever been carried out and the available retrospective studies give an incomplete picture of outcome. It is also difficult to estimate how many patients remain disabled from literature. Hence a number of small studies have been conducted to correlate

particular clinical features with a poor outcome.

Osier and Sidell[59] suggested that patients with a strictly motor neuropathy and no sphincter disturbances were more likely to have a benign prognosis. However Marshall[60] described 37 patients including some with severe sensory loss or sphincter disturbance and could find no evidence that such features influenced outcome adversely. A particularly severe motor deficit appears to carry a greater risk of residual disability according to a number of authors.[61]

Two pediatric [61,62] studies have suggested that the time taken to improve also has prognostic value. They concluded that an interval of greater than 18 days from maximum deficit to onset of improvement (plateau) was associated with incomplete recovery. Other factors common in the poor outcome group were absence of tendon reflexes from onset, severe weakness in distal muscles and a relatively low CSF protein. However majority of studies have failed to find any correlation between CSF protein and outcome.

In a study by J.B. Winer et al,[61] time taken to become bed bound, severity of peak deficit, need for assisted ventilation, age greater than 40, and small or absent compound muscle action potentials in the abductor pollicis brevis elicited by stimulating the median nerve, were found to be associated with poor outcome. However, time from onset of weakness

until improvement began and duration of plateau phase, failed to show a significant correlation with outcome. This conclusion conflicts with previous observations[56] in which failure to improve within 3 weeks of reaching peak deficit adversely affected outcome. In yet another study by NK Singh et al,[63] rapid progression of illness, severe degree of paralysis and muscle wasting, prolonged period of peak paralysis lasting more than weeks, delayed onset of recovery not commencing within 3 weeks from onset of weakness, bulbar paralysis and respiratory involvement adversely affected outcome. Evidence of axonal damage in electro diagnostic studies was also associated with poor outcome. However age, sex, severity of sensory loss, sphincter disturbances, CSF findings and nerve conduction velocity did not significantly affect outcome. Autonomic dysfunctions were noticed in 66.6% cases but were mostly mild and transient, and did not affect long term outcome. Thus in summary, the following factors have been found to be associated with a higher probability of a poor prognosis.

1. **Etiology:** Preceding diarrhea; Campylobacter jejuni infection; presence of GM1 antibodies.
2. **General severity:** Severe, rapidly progressive disease peaking within 7 days from onset; greater maximum disability of arms/legs; early and prolonged (> 1 month) need for ventilatory support;

reduced or inexcitable motor action potential amplitude (< 20% of lower limit of normal).

3. **Absence of immunomodulatory therapy.**
4. **Poor repair:** Old age (> 40 years); longer time until first improvement. However, gender, sensory involvement, sphincter disturbances, CSF protein, CSF cells, all have no effect on prognosis. Autonomic dysfunction is found to occur commonly but mostly mild and transient and does not affect long-term outcome.

# MATERIALS AND METHODS

50 patients diagnosed as Guillain Barre Syndrome (GBS) fulfilling the criteria as modified by Asbury, admitted in the Medical, Paediatric, Neurology wards of Government Rajaji Hospital from January 2009 to March 2011 were included in this study.

**Inclusion criteria:**

Patients fulfilling NINCDS criteria for GB syndrome (Asbury and Cornblath, 1990) and patients with clinical variants of GB syndrome.

**Exclusion criteria:**

Patients with equivocal diagnosis or inadequate clinical details or laboratory investigations were excluded

**Demographic and clinical profile**

Patient's data, type of antecedent event, interval between antecedent event and onset of neurological symptoms, seasonal trends, were recorded in a predesigned protocol (Appendix A).Detailed clinical examination findings, pattern of weakness and sensory and autonomic disturbances, presence of respiratory muscle weakness and requirement on ventilator assistance and mortality were documented. Variants were separated from typical GBS for analysis of clinical details.

**CSF analysis**

Cerebrospinal fluid examination was done in all the patients. The protein level and cell count were looked for albumino cytological dissociation.

**Electrophysiological analysis**

Nerve conduction studies were performed using standard techniques. Motor NCS included stimulation of the median and ulnar nerves at the wrist and forearm while recording from the abductor pollicis brevis muscle and abductor digiti minimi muscle of the hand, respectively, and of the deep peroneal and posterior tibial nerves at the ankle and knee, while recording from the extensor digitorum brevis muscle and the abductor hallicus muscle, respectively. Supraclavicular stimulation was performed on one or more UE motor nerves, usually the ulnar nerve, in an attempt to identify conduction block or slowing proximal to the elbow. F waves were measured with each motor NCS for which a compound muscle action potential (CMAP) result was obtained. The H reflex was recorded from the gastrocnemius and soleus muscles after stimulation of the posterior tibial nerve. The CMAP amplitude, distal motor latency, motor nerve CVs, H reflex amplitude and latency, and shortest F response latencies were measured. Sensory NCS were performed, using antidromic techniques, on the median nerve, ulnar nerve

and sural nerve. SNAP amplitude, latency; conduction velocity were noted

The values were compared with reference values from our laboratory as well as according to Indian standards. Demyelinating neuropathy, axonal neuropathy and conduction block were defined as per criteria recommended by Alpers et al[73] and Oh et al

All patients were treated conservatively with physiotherapy and mechanical ventilatory assistance, where required. Patients who deteriorated in hospital and who could afford it, were treated with intravenous Immunoglobulin (0.4g/kg/for 5 days) and rest of the patients were treated with steroids i.e., intravenous methylprednisolone and plasmapheresis depending on availability of drug and affordability of the patient. Time taken to reach peak deficit, interval from maximum deficit to onset of improvement (plateau time), duration of ventilatory support required and nature of complications were noted.

***During each examination, the following were noted:***

1. **Medical Research Council Grading of muscle weakness.**

    0. No muscle contraction visible

    1. Flicker of contraction but no movement

    2 .Joint movement when effect of gravity eliminated

    3 .Movement against gravity but not against examiner's resistance

4 .Movement against resistance but weaker than normal

5 .Normal power

2. **Hughes Disability Grade (0 - 6) was noted according the following.**

   0 - Healthy.

   1 - Minor symptoms or signs.

   2 - Able to walk 5m without assistance, walking frame, or stick but unable to do manual work including housework, shopping or gardening.

   3 - Able to walk 5m with assistance, walking frame, or stick.

   4 - Chair/bedbound.

   5 - Requiring assisted ventilation (for at least part of day or night).

   6 - Dead.

3. **Bedside Autonomic function tests -** Resting Heart rate, Resting Blood Pressure, Postural Hypotension, Blood pressure changes at the end of one minute and 3 minutes on standing from lying position, wherever possible. In addition complaints suggestive of autonomic dysfunction such as excessive sweating, urinary retention and constipation, palpitations, postural giddiness were also noted.

# OBSERVATIONS AND RESULTS

A total of 50 patients were studied. All patients were hospitalized and the average duration of hospital stay was 17.57 days.

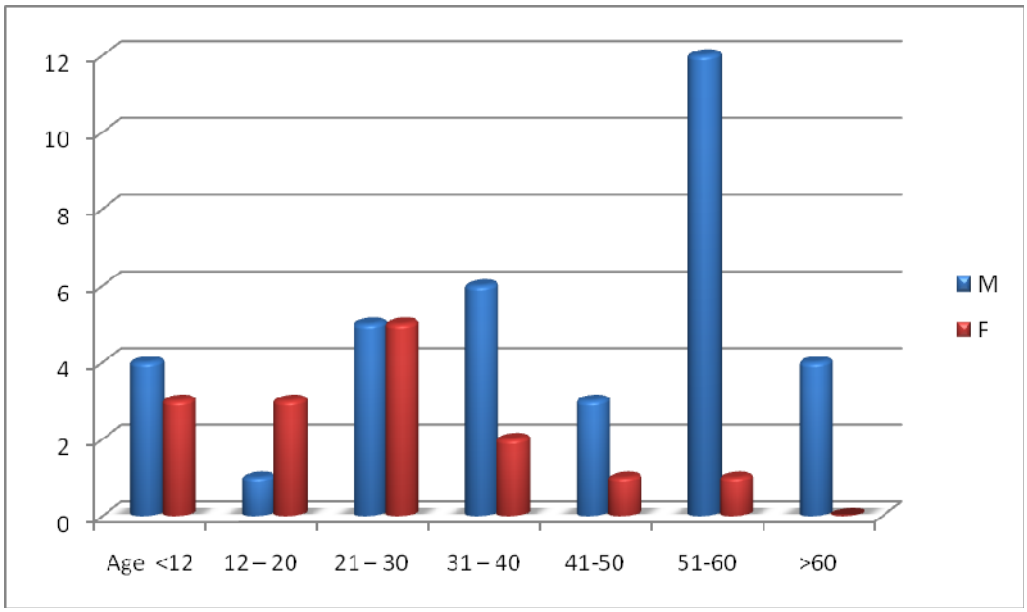
**1. Age and Sex distribution:**

35 patients (70%) were males and 15(30%) were females. The age of patients ranged from 4 to 72 years (Mean age 36.72 years) with the maximum number (26%) of patients in between 51 to 60 yrs age group & in between 21 to 30 (20%) yrs (Table-1).

**Table 1. Age and sex distribution**

| **Sex** | **Age <12** | **12 – 20** | **21 – 30** | **31 – 40** | **41-50** | **51-60** | **>60** |
|---|---|---|---|---|---|---|---|
| **M** | 4 | 1 | 5 | 6 | 3 | 12 | 4 |
| **F** | 3 | 3 | 5 | 2 | 1 | 1 | 0 |
| **Total** | **7** | **4** | **10** | **8** | **4** | **13** | **4** |

# CHART 1. AGE AND SEX DISTRIBUTION



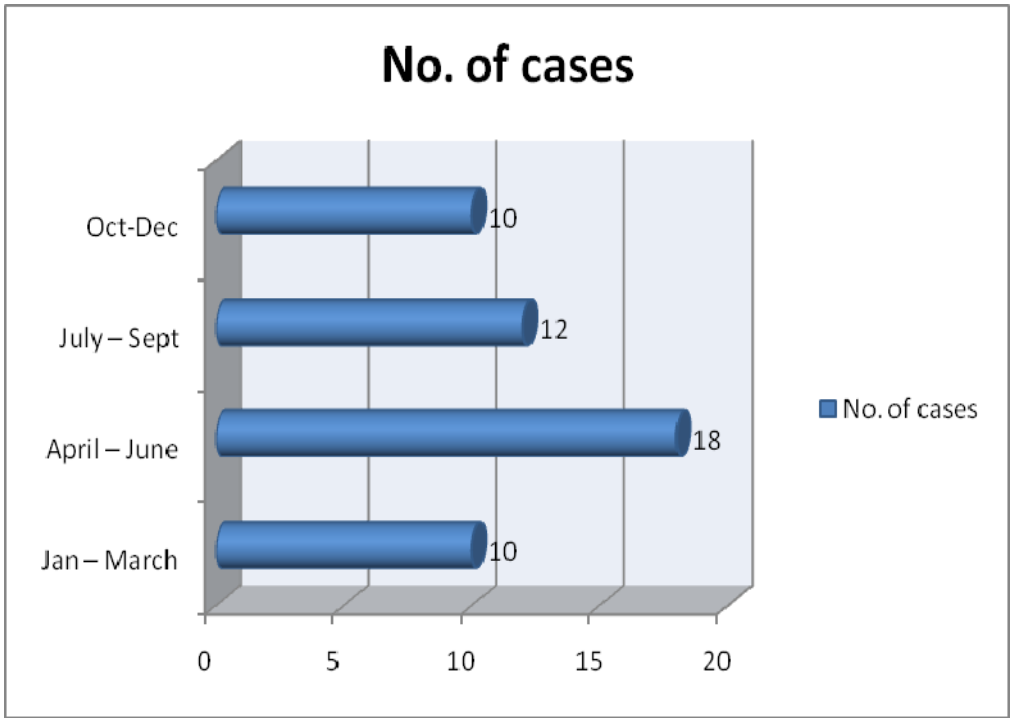
**2. Seasonal incidence:**

Most number of cases were seen in the months of April to June. However no significant increased incidence in any particular season could be inferred. (Table-2).

**Table 2. Seasonal incidence in GBS**

| Months | No. of cases | Percentage |
|---|---|---|
| **Jan – March** | 10 | 20% |
| **April – June** | 18 | 36% |
| **July – September** | 12 | 24% |
| **Oct-December** | 10 | 20% |

## CHART 2. SEASONAL INCIDENCE IN GBS



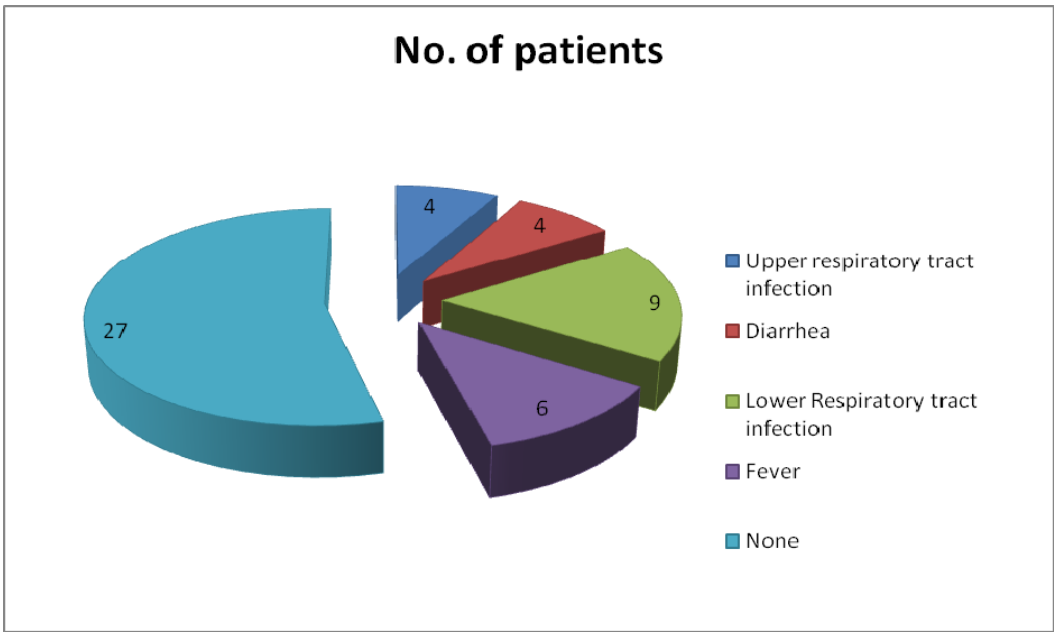
**3. Antecedent illness:**

Twenty three (46%) patients had some antecedent event prior to the development of GBS (Table-3). The most common antecedent illness was lower respiratory tract infection. In patients with a history of preceding illness, the mean duration between onset of GBS and the preceding illness was 9.06 (± 4.21) days.

**Table 3. Antecedent events**

| Antecedent events | No. of patients | Percentage (%) |
|---|---|---|
| Upper respiratory tract infection | 4 | 8 |
| Diarrhea | 4 | 8 |
| Post vaccination | - | - |
| Lower Respiratory tract infection | 9 | 18 |
| Fever | 6 | 12 |
| None | 27 | 54 |

# CHART 3. ANTECEDENT EVENTS



**4. First Symptom of illness:**

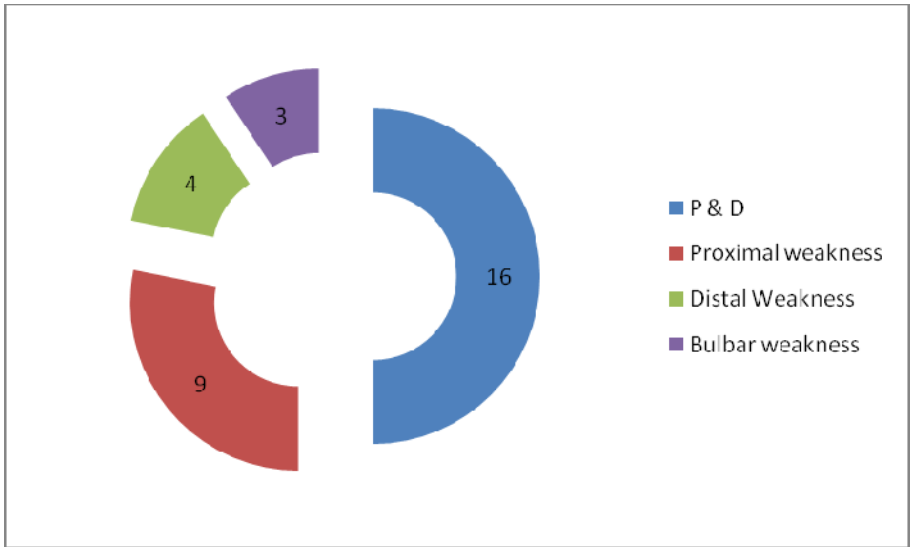
The first symptom of the illness was in the form of motor weakness in 32 (64%) patients and it was sensory in the form of pain, paraesthesia or numbness in the remaining 18(36%) patients. Bulbar weakness was presenting symptom in 3 (6 %) patients and Ataxia in 1 patient.

**Table 4. First symptom**

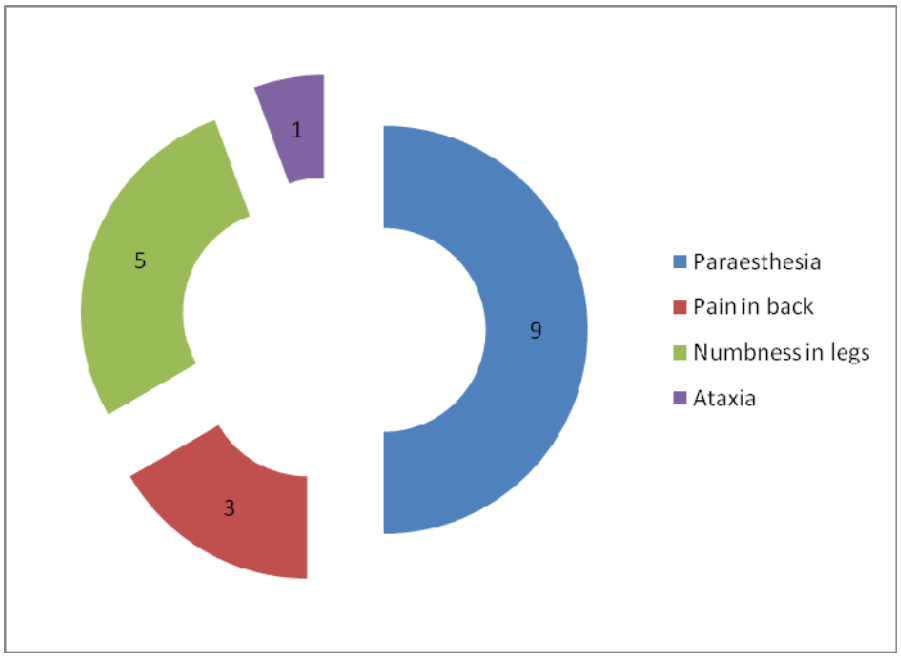
| **Motor symptoms** | **No. of patients** | **Percentage** |
|---|---|---|
| Weakness of UL &LL ( P & D) | 16 | 32 |
| Proximal weakness | 9 | 18 |
| Distal Weakness | 4 | 8 |
| Bulbar weakness | 3 | 6 |
| Total | 32 | 64 |

| **Sensory symptoms** | **No. of patients** | **Percentage** |
|---|---|---|
| Paraesthesia | 9 | 18 |
| Pain in back | 3 | 6 |
| Numbness in legs | 5 | 10 |
| Ataxia | **1** | **2** |
| Total | **18** | **36** |

## CHART 4a . MOTOR SYMPTOMS




## CHART 4b .SENSORY SYMPTOMS



**5. Mode of onset:**

Thirty patients (60%) had ascending form of paralysis. Only 4 (8%) patients had descending type of paralysis (Table-5). Sixteen patients (32%) had simultaneous involvement of both proximal and distal muscles.

**Table 5. Mode of onset of GBS**

| Mode | No. of patients | Percentage |
|---|---|---|
| Ascending paralysis | 30 | 60 |
| Descending paralysis | 4 | 8 |
| Simultaneous involvement of all 4 limbs | 16 | 32 |

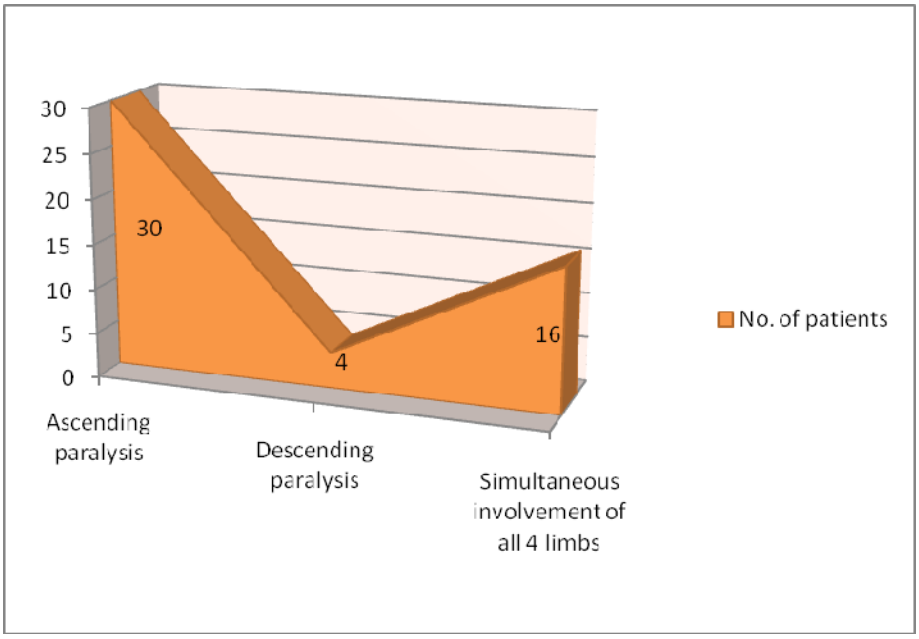
**6. Progression to peak deficit:**

Twelve patients developed maximal deficit in 1 day, majority of patients (48%) developed peak deficit in 2 days.(Table-6).

**Table 6. Progression to peak deficit:**

| DAY(S) | No. of patients | Percentage |
|---|---|---|
| **1** | 12 | 24 |
| **2** | 24 | 48 |
| **3** | 9 | 18 |
| **4** | 4 | 8 |
| **5** | 1 | 2 |

## CHART 5. MODE OF ONSET OF GBS



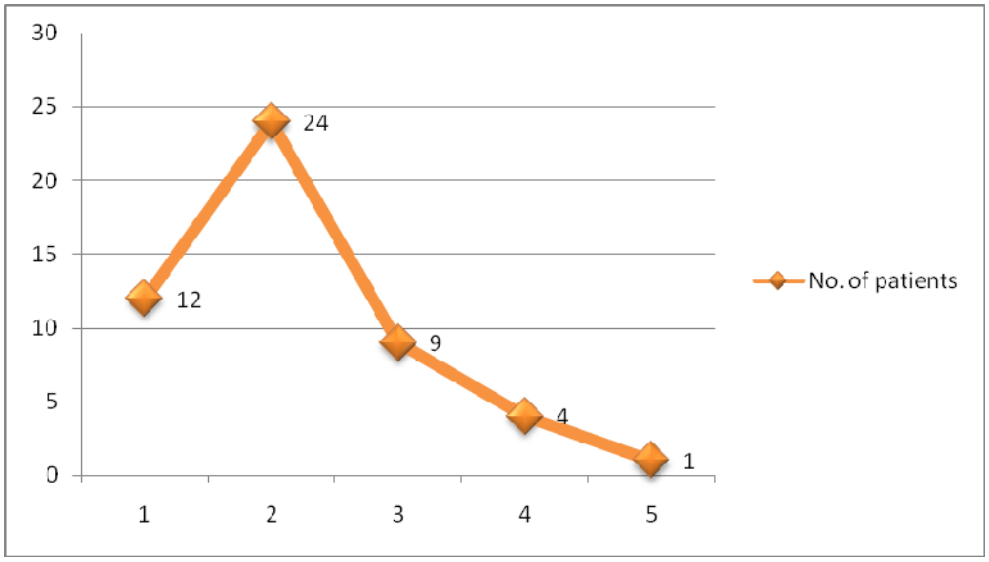
**7. Maximal grades of disability:**

Eight patients (16%) admitted with respiratory distress, Twenty four patients admitted in bed bound state .During the hospital stay, at peak deficit ,Seventeen patients (34%) developed respiratory distress and treated with ventilatory support .Thirty two (64%)patients developed bed bound state during the hospital stay. Three (6%) patients died. The cause of death was respiratory failure following aspiration pneumonia in 1 patient who had rapidly progressed disease. One of the patients, a 17 year old female who had a cardiac arrest, had severe autonomic dysfunction with fluctuating blood pressure and heart rate died on the day of admission itself.

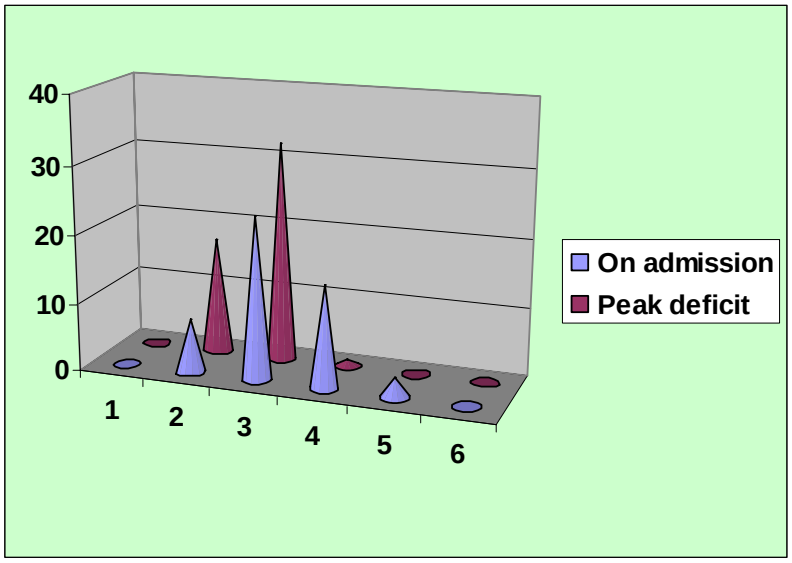
**Table 7. Grade of disability (On admission & at peak)**

| Grade | On admission | | At peak | |
|---|---|---|---|---|
| | No. of patients | Percentage | No. of patients | Percentage |
| **6** | - | - | - | - |
| **5** | 8 | 16 | 17 | 34 |
| **4** | 24 | 48 | 32 | 64 |
| **3** | 15 | 30 | 1 | 2 |
| **2** | 3 | 6 | - | - |
| **1** | - | - | - | - |

## CHART 6. PROGRESSION TO PEAK DEFECIT:




## CHART 7. GRADE OF DISABILITY (ON ADMISSION & AT PEAK)



**8. Sensory Deficit:**

Objective sensory loss was elicited in only 7(14%) out of the 50 patients. The sensory deficit was in the form of diminished vibration and joint position sense, which occurred in a glove and stocking distribution.

**9. Cranial Nerve Dysfunction:**

Twenty eight patients had cranial nerve dysfunction. Twenty seven (54%) patients had facial nerve palsy, among which three patients had unilateral facial nerve involvement which progressed to involve contralateral side also in due course. Nine patients had involvement of 9th and 10th cranial nerves. Total external ophthalmoplegia was observed in two patients. These patients also had severe ataxia, areflexia and weakness in the lower limbs. A diagnosis of Miller Fisher variant of GBS was made in them. One patient had left recurrent laryngeal nerve palsy.

**Table 8. Cranial nerve dysfunction**

| Cranial Nerve | Number of patients | Percentage |
|---|---|---|
| VII – Unilateral, Bilateral | **27** | **54** |
| IX, X | **9** | **18** |
| III, IV, VI | **2** | **4** |

**10. Respiratory muscle weakness:**

Sixteen patients had respiratory muscle paralysis and treated with ventilatory support .The development of respiratory distress is monitored by periodic assessment of maximal inspiratory force and expiratory vital capacity, development of neck muscle weakness, by observing single breath count .

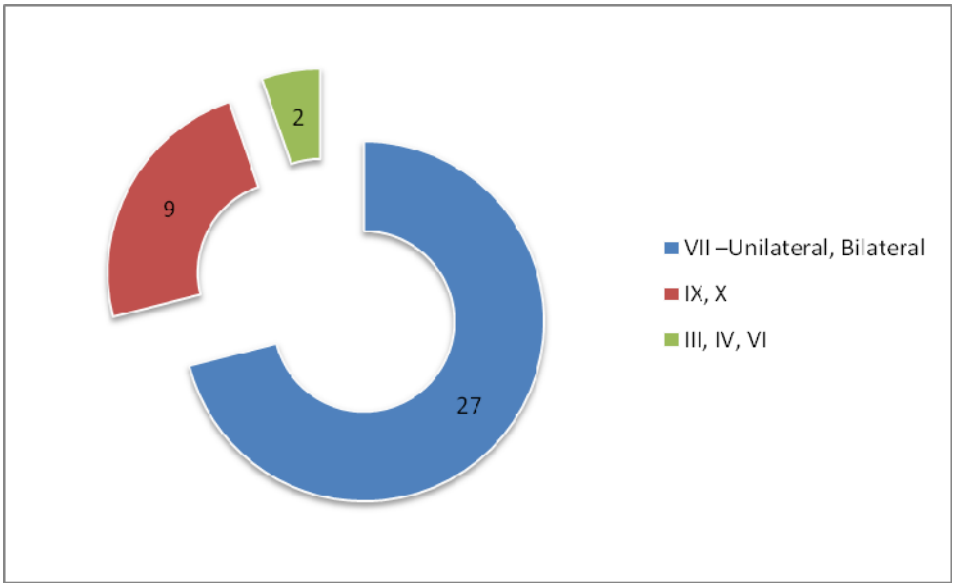
**11. Autonomic dysfunction:**

Patients who were in Grade 4 disability or more were not subjected to standing blood pressure recordings. Only sitting blood pressures were recorded in these patients. In patients who were on ventilator, the spontaneous changes in heart rate and blood pressure were noted. Autonomic dysfunction was detected in 12 (24%) patients (Table.9).

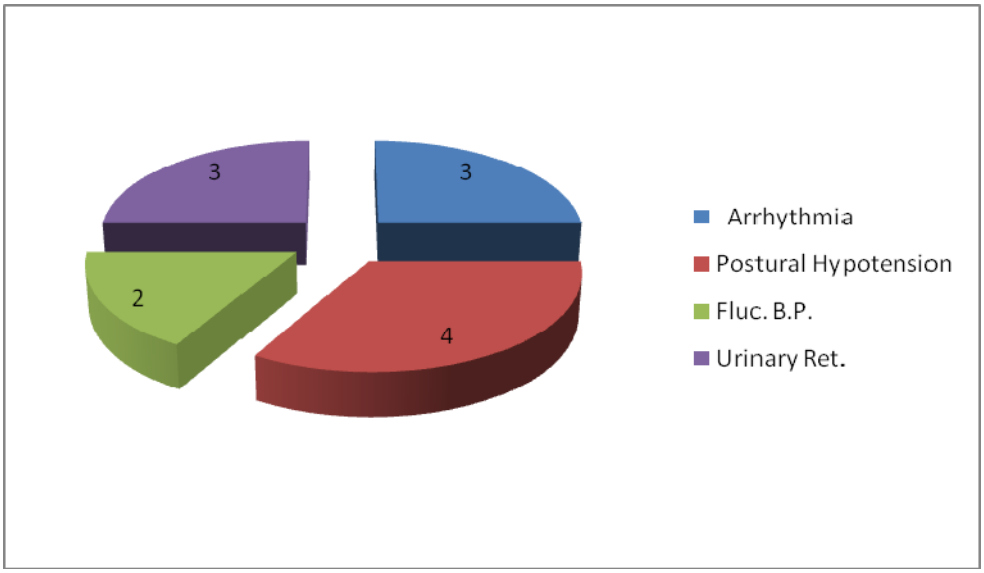
**Table 9. Autonomic dysfunction**

| Autonomic dysfunction | Number of patients | Percentage |
|---|---|---|
| Cardiac Arrhythmia | 3 | 6 |
| Postural Hypotension | 4 | 8 |
| Fluctuating B.P. | 2 | 4 |
| Transient Urinary retention &hesitancy | 3 | 6 |

## CHART 8. CRANIAL NERVE DYSFUNCTION




## CHART 9. AUTONOMIC DYSFUNCTION



**12. Ataxia:**

Two patients who was diagnosed to have Miler-Fisher variant of GBS presented with ataxia. One patient who was admitted in bedridden state, on recovery showed ataxia which recovered subsequently

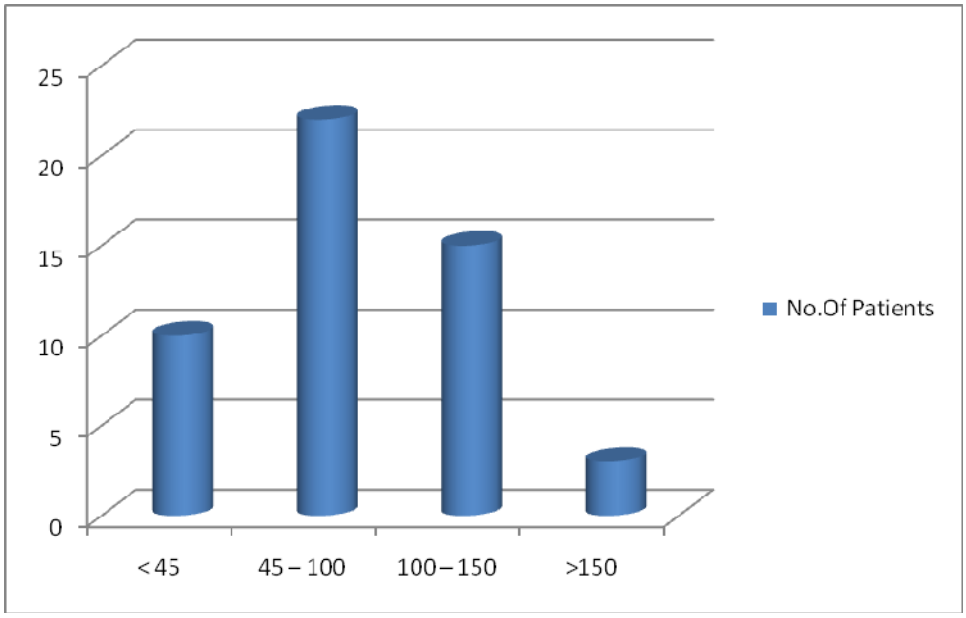
**13. Cerebrospinal Fluid (CSF) Analysis:**

CSF pressure was normal and CSF was clear in all patients. CSF glucose was also normal (approximately half the blood glucose level) in all patients. CSF protein concentration was raised above 50 mg% in 40 (80%) patients at one week. CSF protein level was normal in 10 patients. Three patients had lymphocytic pleocytosis of 20, 30 and 40 cells/cmm. None of the remaining patients had CSF pleocytosis

**Table 10. CSF Protein level**

| CSF Protein (mgms %) | No. of patients | Percentage |
|---|---|---|
| < 45 | 10 | 20 |
| 45 – 100 | 22 | 44 |
| 100 – 150 | 15 | 30 |
| >150 | 3 | 6 |

## CHART10. CSF PROTEIN LEVEL



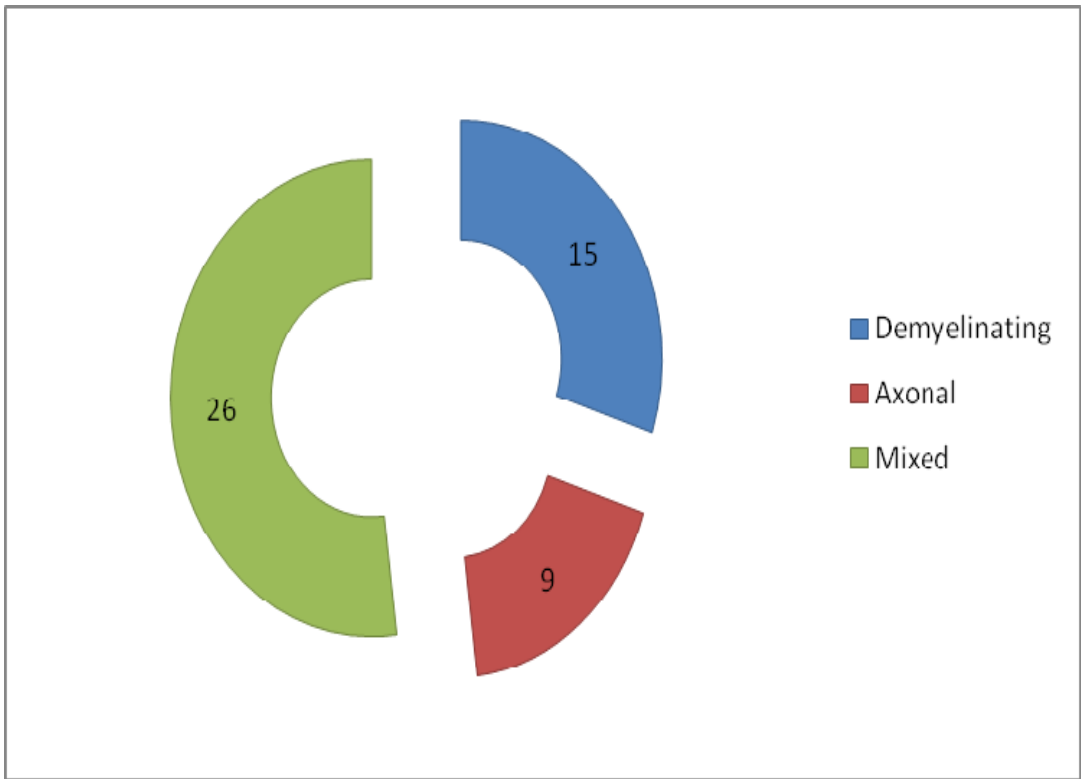
**14. Electrophysiological studies:**

Nerve conduction studies were conducted in all patients. Fifteen patients were found to have reduced motor conduction velocities consistent with demyelinating neuropathy. Nine patients were found to have decreased amplitude of action potentials consistent with axonal pattern of neuropathy. Twenty six patients had mixed pattern of neuropathy. These patients had both demyelinating features (prolonged distal latency, reduced conduction velocity) as well as axonopathy features (reduction of CMAP amplitude)

**Table 11. Nerve conduction studies**

| Type | Number of patients | Percentage |
|---|---|---|
| **Demyelinating** | 15 | 30 |
| **Axonal** | 9 | 18 |
| **Mixed** | 26 | 52 |

**CHART 11. NERVE CONDUCTION STUDIES PATTERN**



**15 .Motor conduction abnormalities**

Motor conduction studies of Median, Ulnar,Peroneal,Tibial nerves were done .Distal latency, distal CMAP amplitude, conduction velocity , H reflex amplitude and latency ,F wave latency were assessed. Varying degree of involvement in these nerves was observed, suggesting multi focal nature of the disease. The H reflex was absent in all cases. The electrophysiological study findings were completely normal, with the exception of absent H waves in 2 patients (4%) in the first week of disease onset.

**Table 12. Motor conduction abnormalities**

**(Percentage of involved nerves)**

| Nerve | DL prolongation | Distal amplitude reduction | CV reduction | Conduction block | Inexcitable nerves |
|---|---|---|---|---|---|
| **Median** | 62 | 80 | 68 | 26 | 10 |
| **Ulnar** | 66 | 75 | 70 | 20 | 8 |
| **Peroneal** | 76 | 66 | 72 | 30 | 26 |
| **Tibial** | 78 | 68 | 76 | 32 | 20 |

**16. Sensory conduction abnormalities**

Sensory conduction studies of Median, Ulnar,Sural nerves were done .distal latency, SNAP amplitude, conduction velocity were assessed. SNAP abnormalities commonly involved in upper limb nerves in the form of conduction velocity reduction and reduced amplitude.Sural nerve less commonly involved .Preservation of sural nerve SNAP confirms the acquired as well as demyelinating nature of the disease in most of the cases

**Table 13. Sensory conduction abnormalities**

**(Percentage of involved nerves)**

| Nerve | CV reduction | Reduced amplitude | Absent SNAP |
|---|---|---|---|
| **Median** | 22 | 20 | 34 |
| **Ulnar** | 26 | 28 | 22 |
| **Sural** | 18 | 18 | 16 |

**CHART 12. MOTOR CONDUCTION ABNORMALITIES**

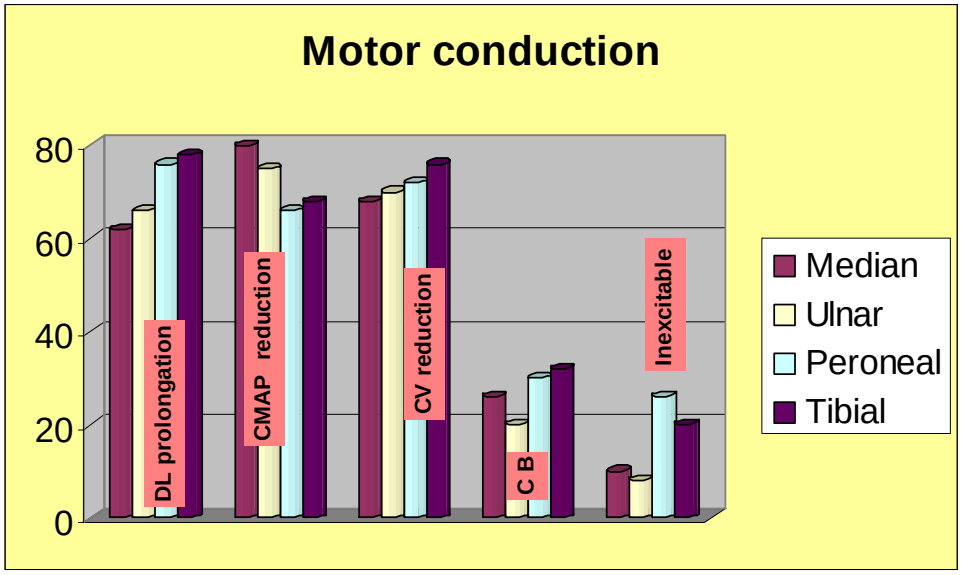
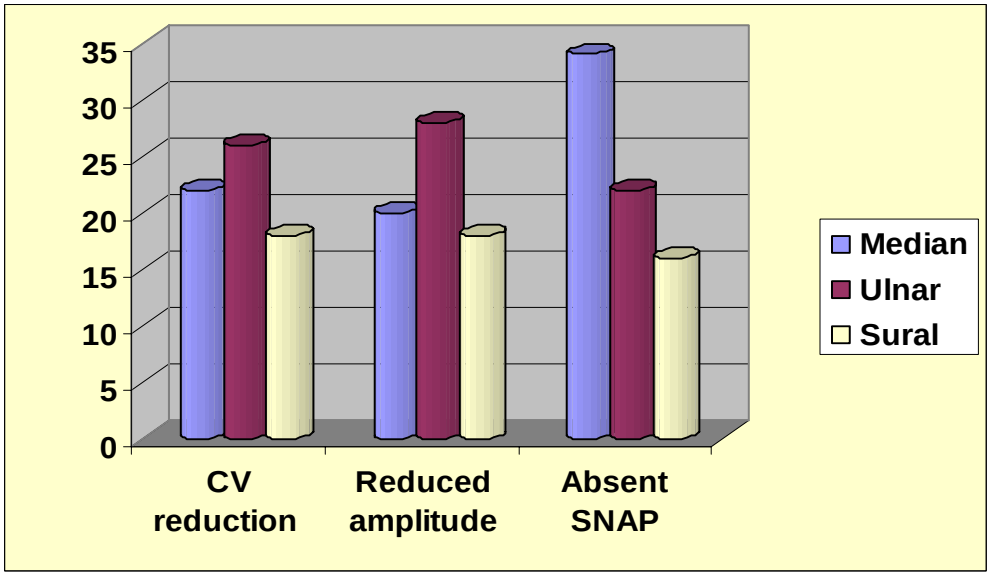



**CHART 13. SENSORY CONDUCTION ABNORMALITIES**



**17 .Complications:**

Aspiration pneumonia is the most common complication in the studied population. Aspiration pneumonia was observed more frequently in patients admitted with bulbar dysfunction .Septicemia occurred in one diabetic patient .Deep venous thrombosis occurred in one patient after prolonged immobilization for which he was treated with low molecular weight Heparin. Urinary tract infection was noted in one patient .E.Coli was grown on urine culture and treated with appropriate antibiotics

**Table 14. Complications**

| **Complications** | **Number of patients** | **Percentage** |
|---|---|---|
| Aspiration pneumonia | 8 | 16 |
| Septicemia | 1 | 2 |
| Urinary tract infection | 1 | 2 |
| DVT | 1 | 2 |

**18. Treatment:**

All patients received physiotherapy and the sixteen patients who developed respiratory failure were put on mechanical ventilation. Two patients received intravenous immunoglobulins in addition to conservative therapy. Both of them showed good response with arrest of progression of muscular muscle weakness by 2 to 3 days. Forty five patients received intravenous methylprednisolone. Patients with intravenous steroids do not show much benefit over supportive treatment. Three patients who were admitted with minimal deficit did not receive steroids.

**Table 15. Various modalities of treatment**

| Treatment | Number of patients | Percentage |
|---|---|---|
| IV MP | 45 | 90 |
| Plasma exchange | - | - |
| IV IG | 2 | 4 |
| No treatment | 3 | 6 |

**19. Mortality:**

Three patients (6%) died in this study. One patient developed aspiration pneumonia and later died due to septicemia and shock. One patient had fluctuating blood pressure and cardiac arrhythmia. She finally died due to cardiac arrest. One patient died of cardiac arrest while on ventilator.

**20. Duration of hospital stay:**

Patients were hospitalized and admitted in various medicine, paediatric wards. Patients who required ventilatory support were transferred to Intensive medical care unit and respiratory support was given. Average duration of hospital stay was 21.44 days .Maximum duration was 29 days in one patient.

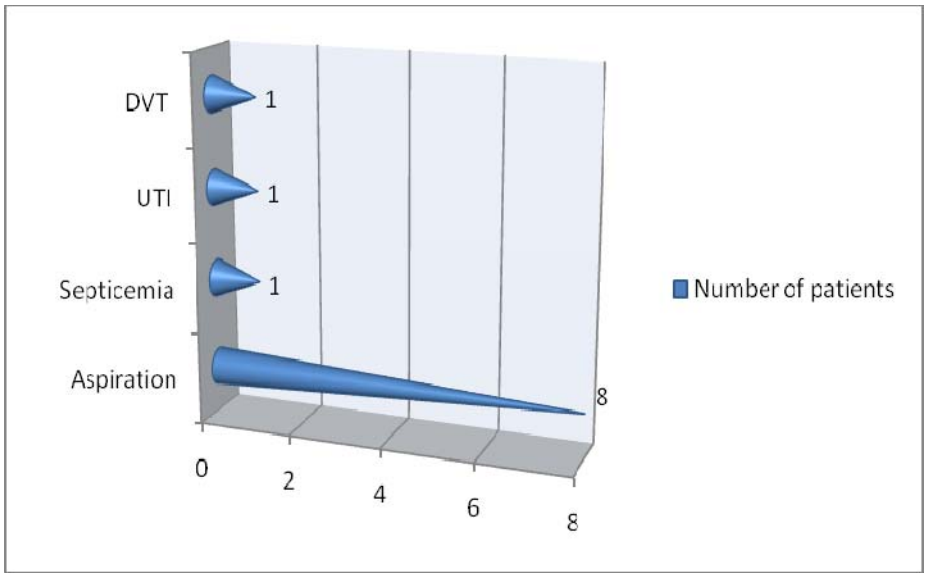
**21. Disability on discharge:**

Disability after discharge was assessed according to Hughes's scale. Most of the patients (64%) were discharged at Grade 3 (i.e. able to walk with support) .Twelve patients (24 %) were discharged at Grade 2.Three patients recovered almost completely; they were discharged at grade 1.

**Table 16. Disability on discharge**

| Grade | No. of Patients | Percentage |
|---|---|---|
| **6** | 3 | 6 |
| **5** | - | - |
| **4** | - | - |
| **3** | 32 | 64 |
| **2** | 12 | 24 |
| **1** | 3 | 6 |

## CHART 14. COMPLICATIONS



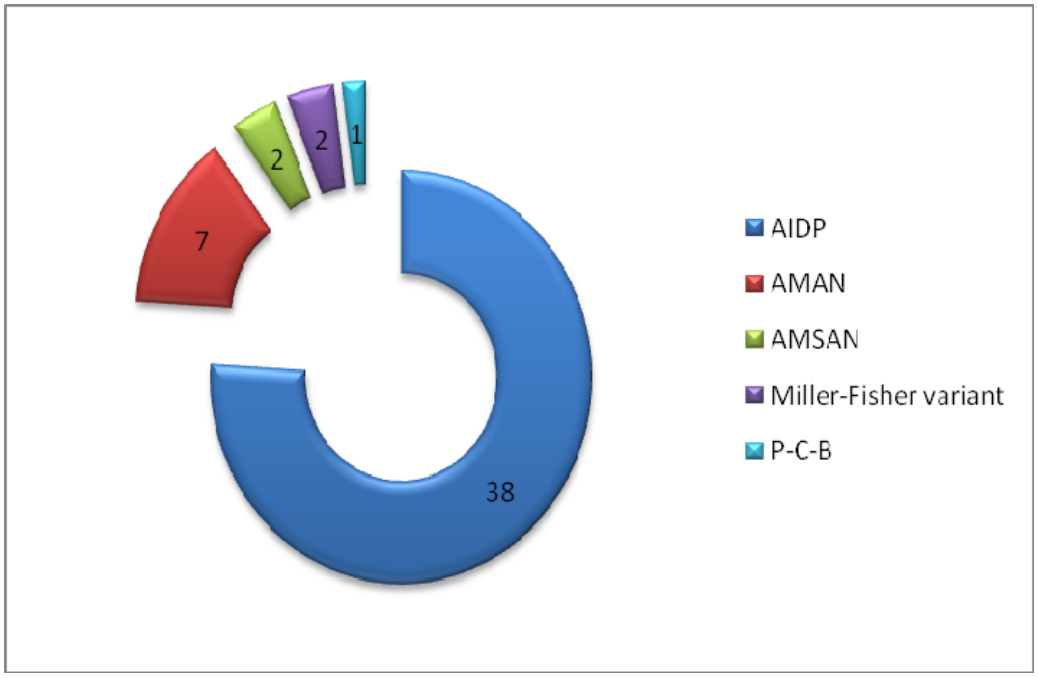
**22. Incidence of subtypes & variants**

Of 50 cases of GBS ,acute inflammatory demyelinating neuropathy (AIDP) was the most common subtype forming 38 cases (76%) followed by Acute motor axonal neuropathy (AMAN) **.**Acute Motor Sensory Axonal Neuropathy was observed in 2 (4%) patients. Miller – Fisher variant of GBS was observed in 2 young male patients. One patient presented with Cervico-brachial-pharyngeal variant..

**Table 17. GB syndrome variants**

| Variants | No. of patients | Percentage |
|---|---|---|
| **AIDP** | 38 | 76 |
| **AMAN** | 7 | 14 |
| **AMSAN** | 2 | 4 |
| **Miller-Fisher variant** | 2 | 4 |
| **Cervico-brachial-pharyngeal variant** | 1 | 2 |

## CHART 16. GB SYNDROME VARIANTS



# DISCUSSION

A total of 50 patients were included in this prospective study. The maximum number of patients was in between 51 to 60 years age group (26%). Kaplan et al[16] reviewed 2575 cases and found the peak incidence to be between 50 and 74 years of age with lesser peak between 15 and 35 years. Similarly Peter C. Dowling[65] also reported two peaks. In Thakaran et al[50] series however, the mean age of study group was only 28 years.

There is a male preponderance in our study which is in conformity with the report by Robert M. et al.[66] However, Peter C. Dowlin's[65] study of 176 patients Showed an equal incidence in males and females.

No seasonal variation in incidence of GBS could be inferred from this study in conformity with the majority of studies in literature[66]. However a few studies have noted a seasonal clustering of cases. Kaur et al[67] reported an increased incidence in summer and autumn. Peter C. Dowling[65] also noted an increase in summer.

Twenty three (46%) of our patients had a definite antecedent event prior to onset of illness. Winer et al[61] reported that over half of GBS patients experience symptoms of viral respiratory or gastrointestinal

infections. Ropper et al also reported a high incidence of 73%. In contrast a study by Kaur et al[67] showed a lower incidence of 32%. Zhahirul Islam et al showed 69% had antecedent illness of which 37% of cases were preceded by diarrhoea[71].

The interval between prodromal illness and onset of GBS is most frequently from 1-3 weeks. Occasionally it is as long as 6 weeks. Kaur et al[67] reported a mean interval of 9.2 days. In our study there is a mean interval of 9.06 (± 4.21) days between the prodrome and the onset of GBS. The most common antecedent illness was lower respiratory tract infection (18%) while diarrhoeal illness and upper respiratory tract infection was seen in four patients (8%).

Ascending paralysis was noted in 60% (30 patients) and descending paralysis in 8% (4 patients), while 32 %( 16 patients) had simultaneous involvement of all four limbs. According to description by Winer et al[61] that muscle weakness usually starts in legs and ascends to arms in most cases. A metaanalysis of large series by Allan H. Ropper[21] showed ascending paralysis in 60%, descending paralysis in 20% and involvement of all four limbs simultaneously in 20% cases.

In 32% patients, the first symptom of illness was motor in the form of flaccid paralysis of both upper and lower limbs, 34% had paraesthesia in hands and feet, numbness or pain, bulbar symptoms was present in

6%of cases. One patient presented with ataxia. However Robert et al reported first symptom as sensory in 83% and motor in 17% of patients. Allan H. Ropper[21] in his metaanalysis reported 85% incidence of paraesthesia. In a study by Winer et al61 75% patients had paraesthesia, Robert M, et al described 83% incidence in paraesthesia. Objective sensory loss occurred in 7 patients (14%) in the form of diminished touch, vibration and joint position sense which occurred in glove and stocking distribution. This is much lower than the 40% reported by Allan H. Ropper [21] in his metaanalysis. Winer et al [61] noted sensory loss in 52% of his patients.

All patients had involvement of the legs and involvement of limbs was symmetric in all cases. None of the patients had involvement of hands alone, which is in conformity to the observation of Winer et al[61] who said that the arms are not affected in isolation. Two patients also had ataxia and involvement of 3$^{rd}$, 4$^{th}$ and 6$^{th}$ cranial nerves. They were diagnosed to have Miller Fisher Variant of GBS.

Respiratory failure was present in 32% of our patients. Allan H.Ropper [21] in his meta analysis showed that 10% of patients have respiratory failure. Winer et al[61] noted a 23% incidence of respiratory failure. The average duration of mechanical ventilation in our patients was 9.12 days.

Twenty four patients (48%) presented with bedridden state, eight patients(16%) presented with features of respiratory distress and immediately needed ventilator support ,seventeen patients (34%) reached grade V disability and Thirty two (64%) patients reached grade IV (bedridden state). In the study by Winer et al[61] only 12% retained the ability to walk throughout the illness and the remaining 88% became bedridden. This is in contrast to the report by RDM Hadden et al[58] who said 40% patients become bedbound.

Overall, about 50% of patients with GBS reach maximal weakness by 1 week, 70% by 2 weeks, and 80% by 3 weeks in the course of illness[61]. In this study 24% of patients reached peak deficit within 1 day of onset of illness, 90% by 3 days.

56% of our patients had cranial nerve dysfunction.20% of patients had involvement of multiple cranial nerves. This is in conformity with the 50% incidence reported by Winer et al[61] and 60% in Allan H. Ropper's[21] meta analysis. Kaur et al[67] reported an incidence of 41% in her study from North India.

Facial Nerve was the most commonly involved 27 patients in our series in concordance with most series. IX and X cranial nerves were involved in only 9 patients in contrast to the reported incidence of 50% in Allan H. Ropper's meta analysis.3,4,6 cranial nerves involved in 2

patients. Other cranial nerves including recurrent laryngeal nerve was involved in 2 patients

Autonomic dysfunction is reported to occur in up to 50% of GBS patients P.Hachenecker et al[68] noted dysfunction in 69% of their patients. NK Singh et al[63] documented 67% incidence. In this study autonomic dysfunction occurred in 24% of patients. Cardiac arrhythmia occurred in 6% of cases, postural hypotension in 8% of cases, Fluctuating Blood pressure was noted in 4% of patients. Transient sphincteric dysfunction in the form of urinary retention and hesitancy was seen in three (6%) patients. Allan H.Ropper's meta analysis reported 15% incidence of transient bladder disturbances in GBS patients NK Singh et al[63] observed sphincter disturbance in 20% of patients.

CSF protein was raised above 50mg% in 40 patients.Winer et al[61] reported raised CSF protein in 80% patients while 90% was reported in Allan H.Ropper's[21] meta analysis. The lower number of patients with raised CSF protein in this study was probably because all CSF studies were done between 1 and 2 weeks from onset and not repeated thereafter. It is possible that there may have been a rise in CSF protein later in the course of illness, which was not recorded. Furthermore, it has been noted in some studies that CSF protein may not rise throughout the course of

illness in some patients with GBS. Gupta RC[63] reports such patients did not show rise in CSF protein even at 6 weeks.

CSF pleocytosis was seen in three patients. CSF mononuclear cell counts of up to 50 per cmm may be seen in GBS and does not rule out diagnosis of GBS.

Electrophysiological studies were conducted in all patients and 15 of them showed demyelinating pattern, 9 of them showed axonal pattern, 26 patients mixed pattern .Patients having mixed and axonal pattern showed poor prognosis compared to patients having demyelinating pattern. Two patients showed normal conduction study initially but repeat nerve conduction showed demyelinating pattern, and two patients initially had prolongation of f latency and absent H wave as the only feature and rest of the conduction were normal , on repeat conduction after one week showed demyelinating pattern

Many authors have found a proportion of patients to have normal nerve conduction and also involvement of nerves in varying severity. The population varies form 9% to 20%.[70] and is higher in the first few weeks of illness. This multi focal involvement has been explained as due to

1) The patchy nature of pathology of GBS which means that studies confined to one or two nerves may miss abnormal findings. 2) Maximum conduction velocities may conceal abnormalities since

conduction can occur normally in some fibres while being partially blocked in some others. 3) Lastly it is likely that proximal conduction blocks occur commonly in GBS that distal motor conduction would be unaffected[76]

Three patients died in this study. One patient developed aspiration pneumonia and later died due to Septicemia and shock. One patient had fluctuating blood pressure and cardiac arrhythmia and died due to cardiac arrest .one patient died due to cardiac arrest while on ventilator.

Case fatality in this study was 6%. Mortality in GBS varies from 1.3% to 13% in different series with a mean of about 6% Winer et al[61] reported 13% mortality in his study of 100 patients. NK Singh et al[63] noted 8% mortality.

Total duration of hospital stay varied from 29 days to 16 days, average being 21.44 days. Most of the patients were being discharged at grade 3(64%), 24% cases discharged at grade 2, 6% cases were recovered almost completely from the illness.

Of all GB syndrome variants AIDP subtype predominates which was demonstrated in various studies. In this study 38 patients (76%) diagnosed to have AIDP, 7 patients (14%) diagnosed to have AMAN,2 patients (4% ) diagnosed to have AMSAN. Other variants like Miller-Fisher variant was observed in 2 patients and Cervico pharyngeal

brachial variant was observed in 1 patient.

AIDP is the predominant subtype in United states and Europe (up to 90%) and axonal subtype predominates in china (70 % AMAN, 25%AIDP,5% others) [73] .Hadden etal[74] noted 71% AIDP,4%AMAN,2%AMSAN,1% Miller Fisher subtypes in his study. Zhahirul Islam[71] et al showed AIDP in 82% cases and AMAN in 15% cases. Gupta et al noted AIDP in 70% cases, AMAN in 20% cases, Miller Fisher variant in 5 % cases[72] in India.

# SUMMARY

In this prospective study of 50 patients with GBS (based on Asbury's Criteria), it was found to be commonest in the age group above 50 years and there was a male preponderance.

Consistent with the known features of GBS, no significant seasonal predilection was present and 46% of the patients gave a history of a definite antecedent illness prior to the onset of GBS. The most common antecedent illness was lower respiratory tract infection seen in 18% of our patients followed by a non descript fever in 12%, followed by upper respiratory tract infection and gastro-intestinal illness

Areflexic symmetric motor paralysis was the presenting feature of all the patients with the majority showing ascending type of paralysis (60%).The onset of GBS was heralded by both motor and sensory symptoms. Acute onset of flaccid weakness of both upper and lower limbs as well as predominant proximal weakness and sensory symptoms in the form of paraesthesiae confined to the fingers and toes were the common presentation.

Thirty two (64%) reached bed bound state with 16

patients (32%) required ventilatory support. Seventy two percent of patients reached maximal disability by 2 days, 90% by 3 days and 98 % by 4 days of onset of illness

Autonomic dysfunction was seen in 24% of patients and was mostly mild and transient. However severe autonomic dysfunction was seen in one patient and was the cause of his death due to cardiac arrhythmia. Sphincter dysfunction in the form of urinary retention and hesitancy was transient, lasting 2 – 3 days and occurred in6 % of patients.

Cranial nerve dysfunction occurred in 56% of patients with facial nerve being the commonest nerve involved (54%).20% of patients suffered with multiple cranial nerve palsies.

Contrary to most reports, which state up to 40% objective sensory loss, we noted objective sensory loss in only 14% of our patients

CSF protein was raised above 45mg% in most of the patients (80%), after 1 week of illness.CSF cell count showed reduced cell count demonstrating albumino cytological dissociation.CSF lymphocytic pleocytosis was seen in 3 patients. However, all 3 patients fell well within the modified Asbury's criteria for diagnosis of GBS which states that a pleocytosis of up to 50 cells / cmm may be seen in patients of GBS.

Electrophysiological studies conducted in all patients revealed demyelinating pattern in 15 patients, axonal pattern in 9 patients and mixed pattern in 26 patients.

AIDP was the most common variant in studied population .Seven patients diagnosed to have AMAN variant, two patients diagnosed to have AMSAN variant. Two patients who are presented with ataxia later had ophthalmoparesis and diagnosed as having Miller Fisher variant .One case of Cervico-brachial-pharyngeal variant also evaluated in our study.

Patients with AMSAN type developed muscle wasting later and had permanent disability after 3 months of follow up .All the three patients who died were diagnosed to have AIDP.But there is no definite correlation exists between the type of presentation with fatality and disability.

# CONCLUSIONS

1. GBS occurs in all age groups with a greater incidence in the older age group above 50 years. However age did not have any correlation with prognosis.

2. GBS affects both sexes; however males were affected more than females in the ratio of 7:3 in this study.

3. Nearly 1/2 of patients reported a definite antecedent event prior to onset of GBS.

4. Onset of GBS is heralded by both motor and sensory symptoms However; objective sensory deficit is seen in very few patients.

5. Ascending type of paralysis was most commonly seen in our study.

6. Progression to maximal motor deficit occurred within 3 days in 90% of patients. Progression of muscle weakness beyond 4 weeks is not seen.

7. Cranial nerve dysfunction occurred in 56% of patients in GBS. Facial nerve was most commonly involved.

8. Autonomic dysfunction occurred in 1/4$^{th}$ patients.

9. Respiratory failure occurred in 1/3rd of patients in GBS.

10. Albuminocytological dissociation was seen in majority of patients of GBS after 1 week. However, CSF protein level has no prognostic value.CSF protein level may be normal in some cases initially, repeat studies may show elevated protein levels.

11. Rapid progression from onset to peak paralysis, prolonged duration of peak paralysis, need for ventilatory support and severity of paralysis were the factors associated with poor recovery from the illness.

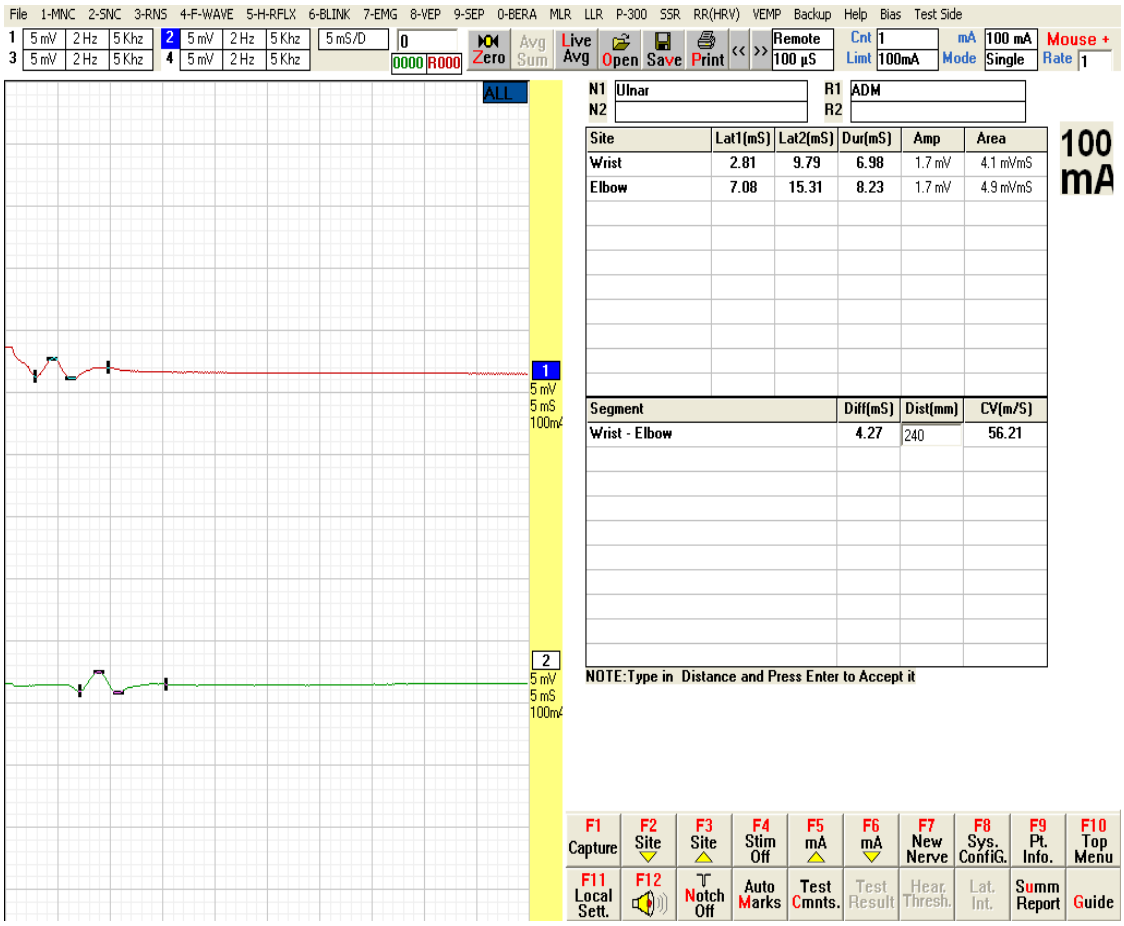
12. Demyelinating with secondary axonal neuropathic pattern (Mixed pattern) was the commonest electro physiological abnormality in this study.

13. Conduction abnormalities were not similar in all the nerves studied. Varying severity of involvement may occur in nerves due to multi focal demyelination

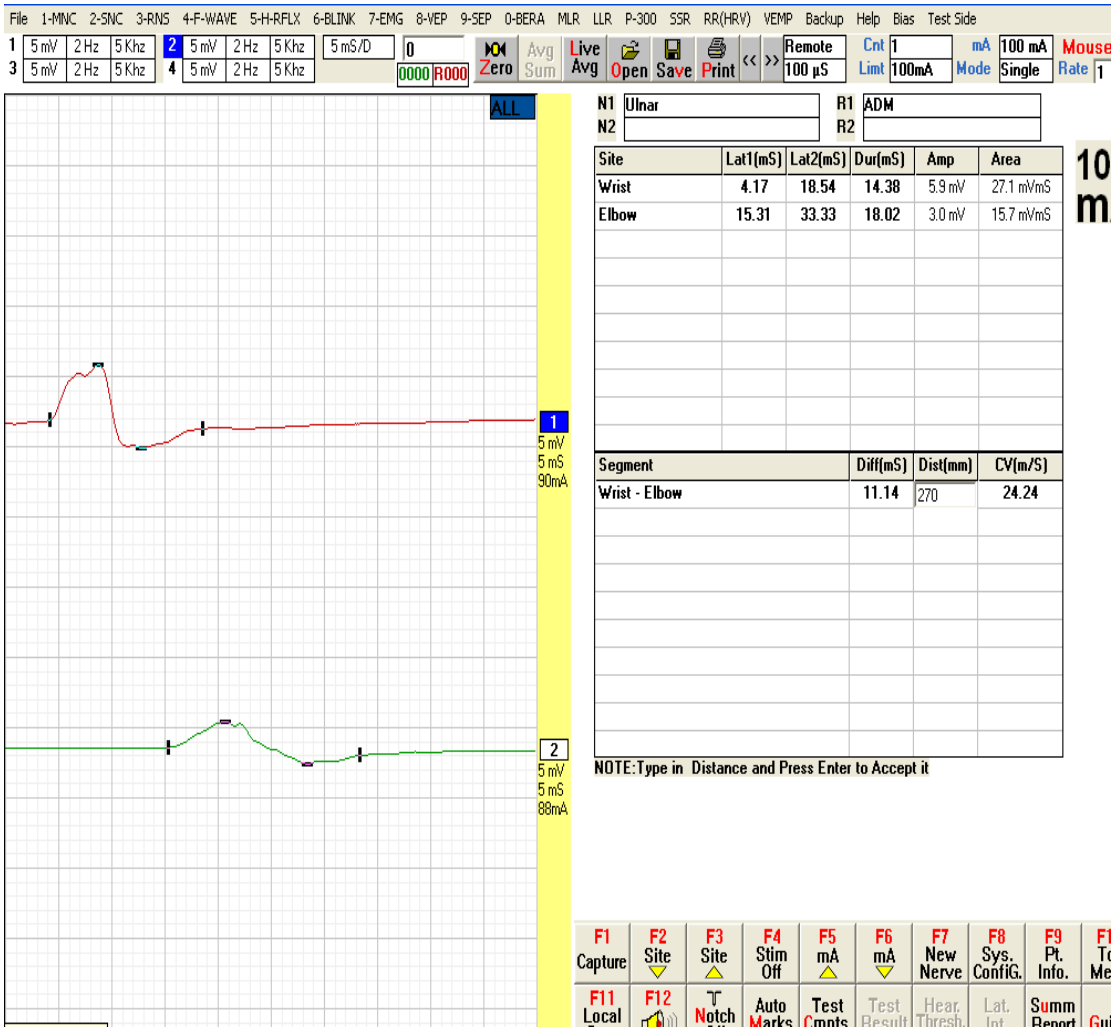
14. Mortality in GBS was 6% in our study.

15. AIDP was the commonest subtype in studied population, followed by AMAN variant

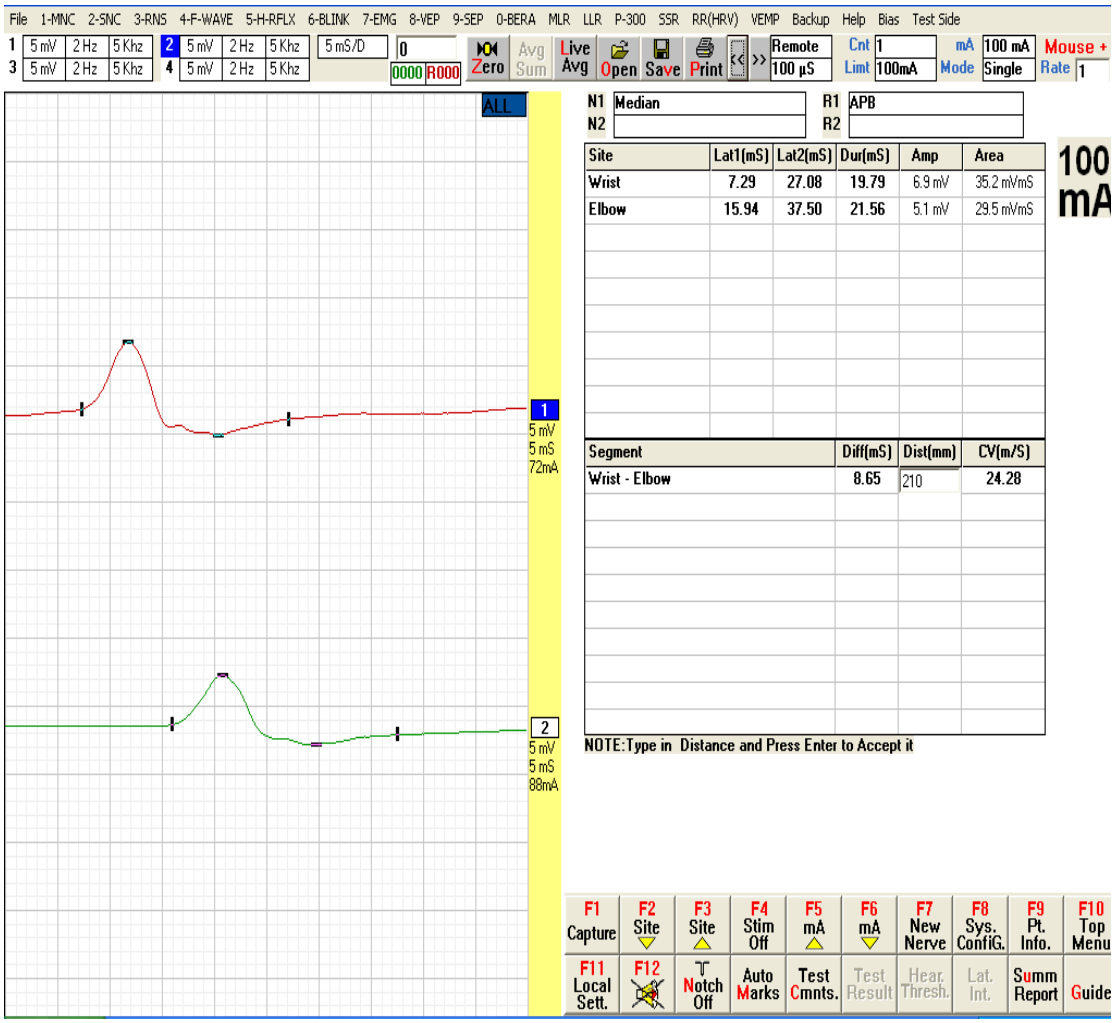
# AXONAL NEUROPATHY OF ULNAR NERVE



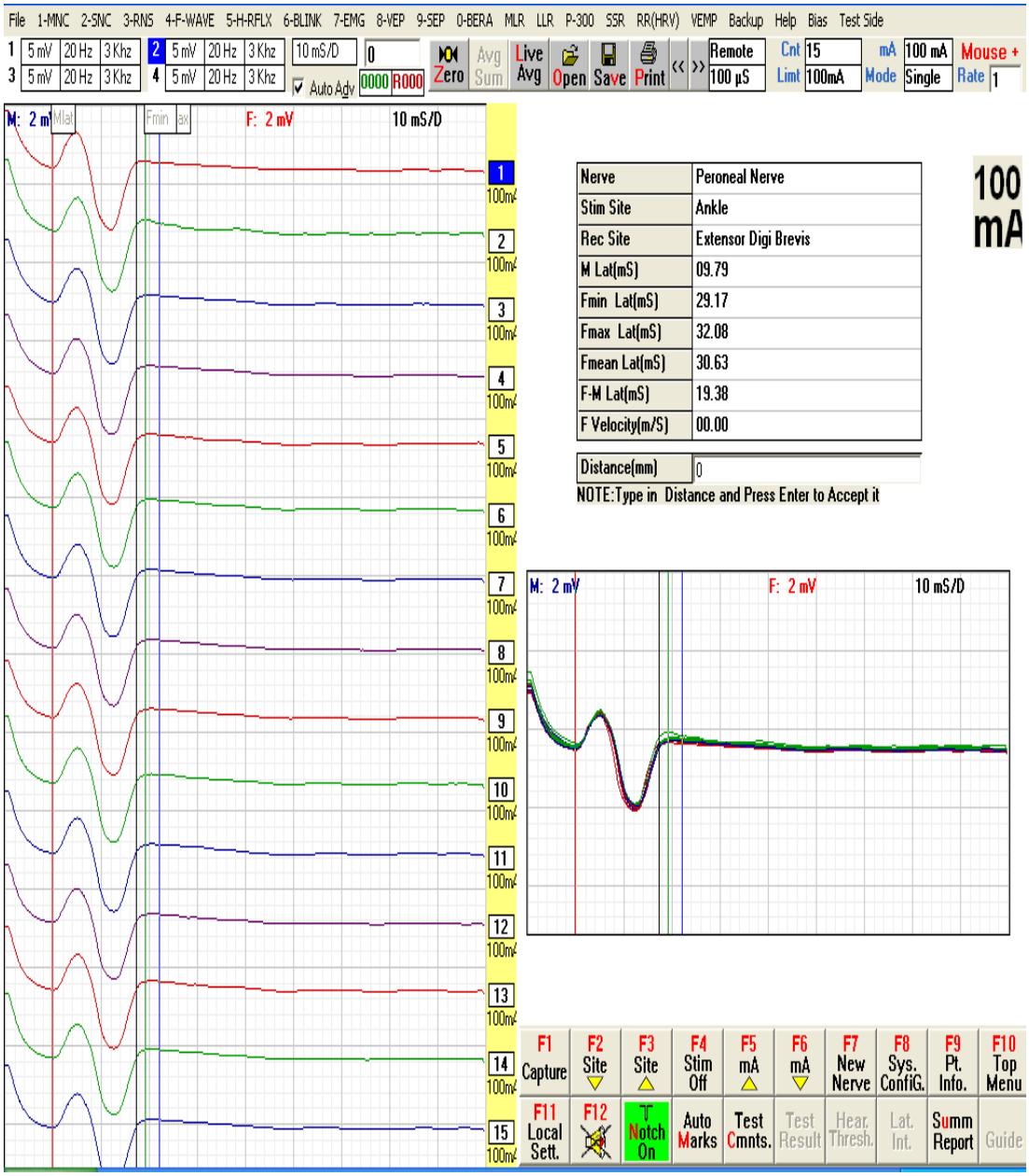
# ULNAR NERVE SHOWING CONDUCTION BLOCK



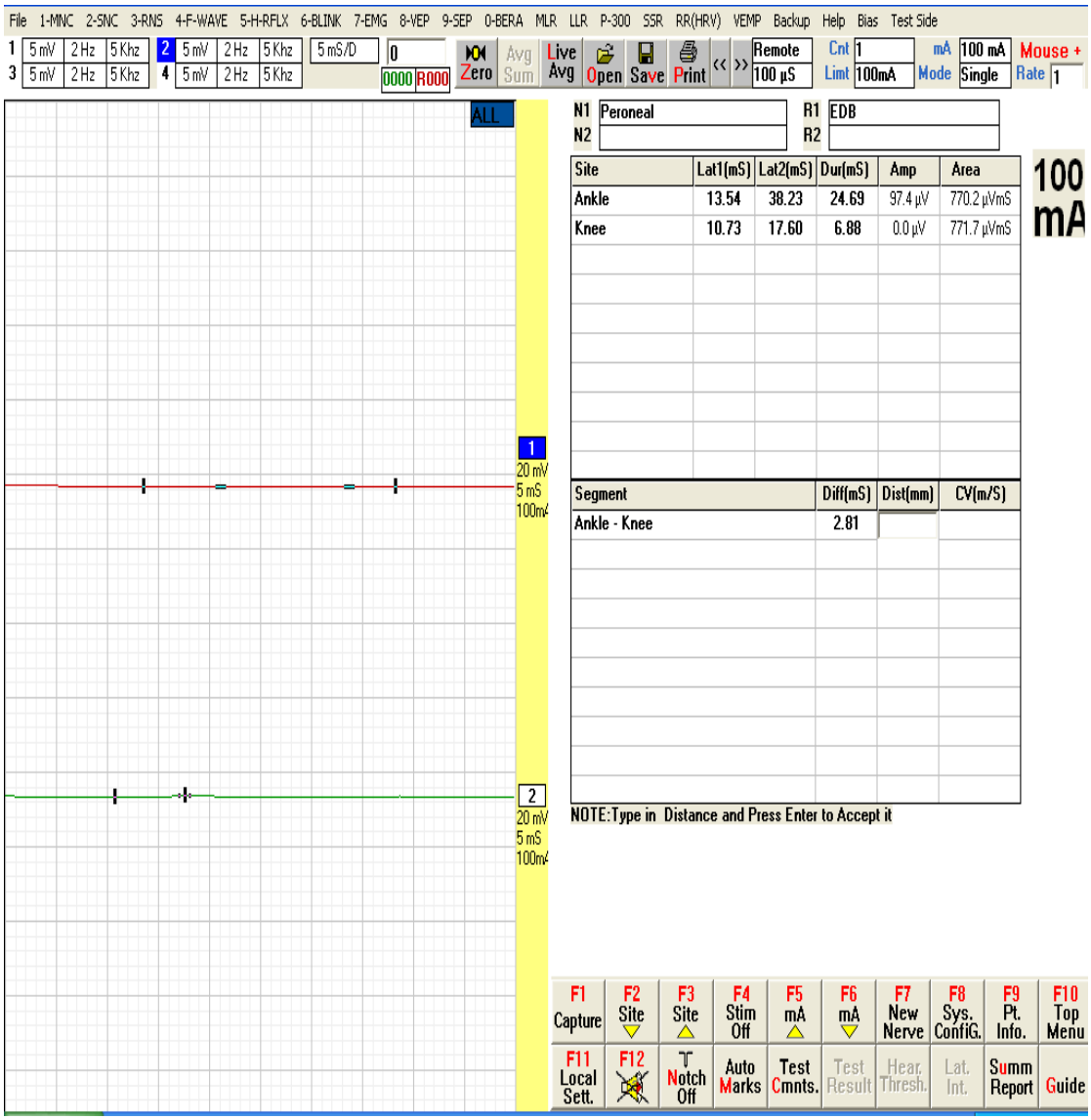
# DEMYELINATING NEUROPATHY OF MEDIAN NERVE



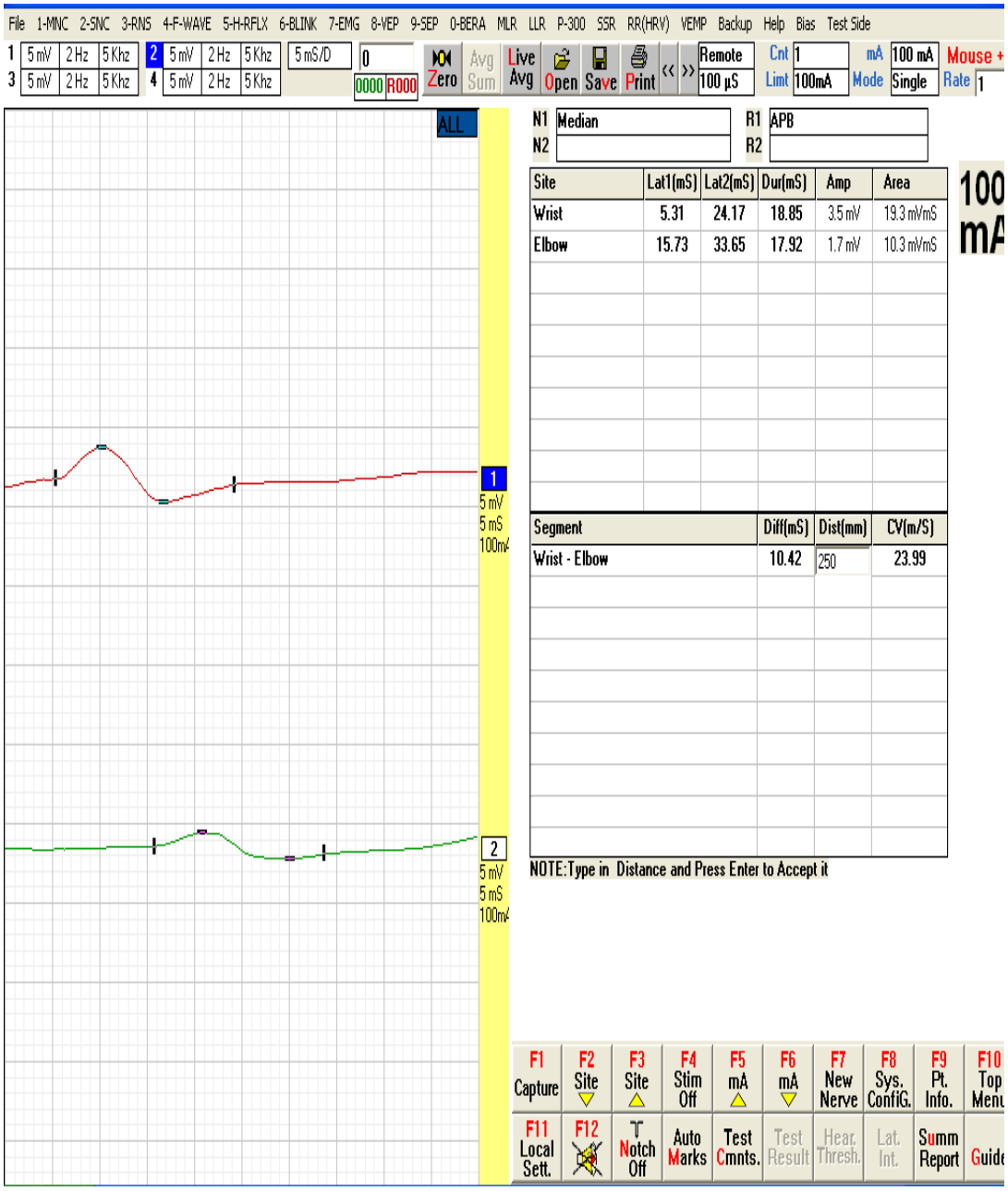
# ABSENT F WAVES



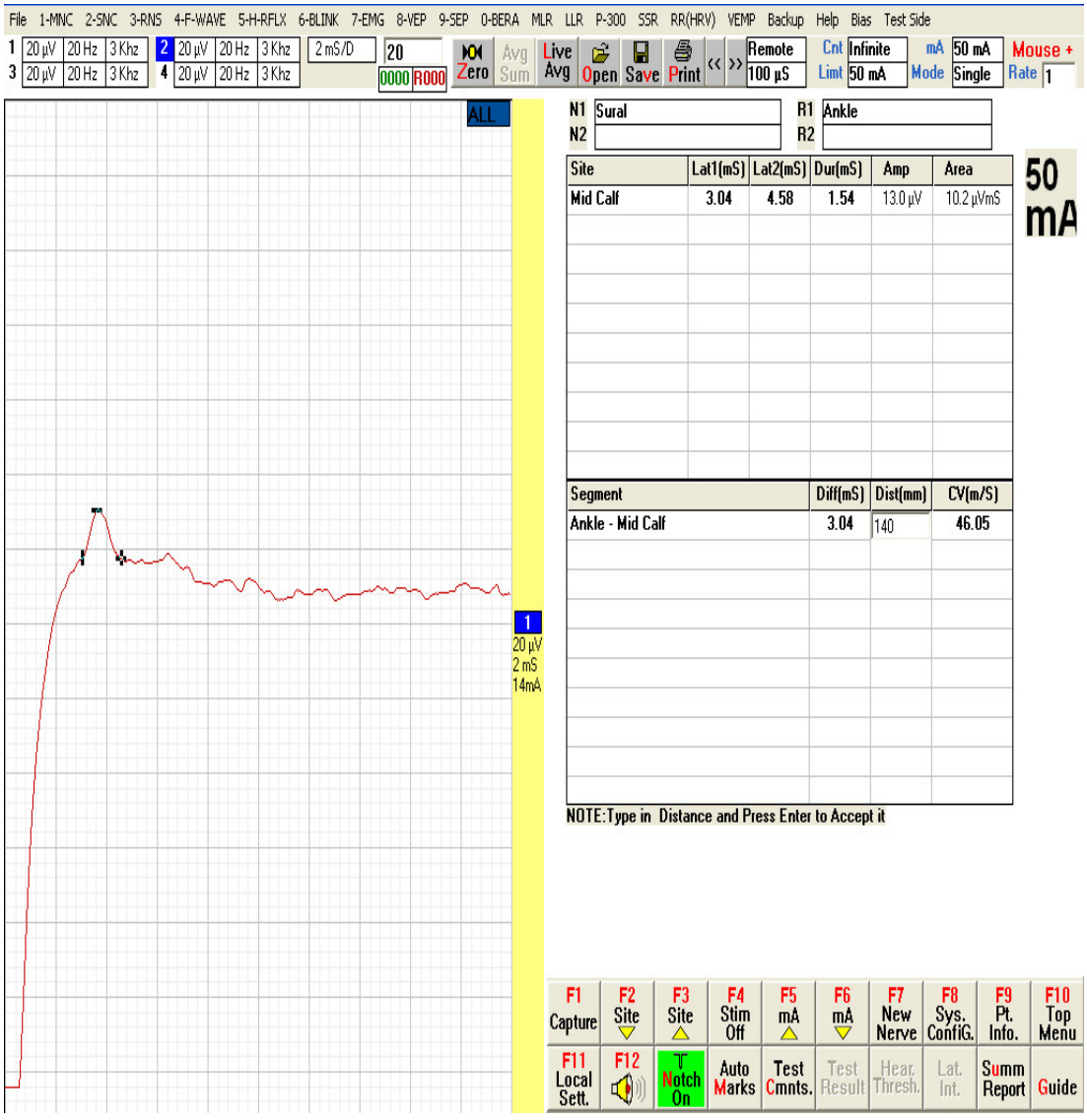
# INEXCITABLE NERVE – PERONEAL NERVE



# MIXED PATTERN IN MEDIAN NERVE



# SURAL NERVE –NORMAL SNAP



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**PROFORMA**

Name: ---------- Age / Sex: ------- yrs /

I. P. No: ---------- Unit: ----------

Date of Admission: ---------- Languages Spoken: ----

Date of Discharge: ---------- Economic Background: -

Handedness: ---------- Level of Education : ------

**DIAGNOSIS:**

Present History:

Time of Onset: duration:

Progression of the disease:

Weakness of Right UL Left UL Right LL Left LL

Pattern of weakness:

Predominant proximal / Predominant distal/ Mixed

Facial weakness: Yes/No

Bulbar dysfunction: Yes/No

Other Cranial nerve dysfunction: Yes/No

Neck muscle weakness: Yes/No

Respiratory difficulty: Yes/No

Sensory symptoms: Yes/No

Autonomic dysfunction: Yes/No

Unsteadiness of gait : Yes/No

Preceding H/O Fever ( )

Diarrhea ( )

Respiratory tract infection ( )

Exanthematous illness ( )

Past History:

Systemic illness (HTN, DM, IHD, any other systemic illness):

Medication:

Insecticidal exposure:

Dog bite /snake bite:

Vaccination:

Other treatment History:

Personal History:

H/O venereal exposure
Smoking
Alcohol
Diet

Family history:

## General Examination

Anaemic/jaundice/clubbing

Generalized lymphadenopathy

Peripheral nerve thickening

Neuro Cutaneous markers

Alopecia/joint swelling/Rashes

Vital Parameters:

BP: Right arm mm of Hg

Left arm mm of Hg

Pulse:--------per min; regular/irregular

Peripheral Pulse:

Carotids:

Respiration Rate & Rhythm

Chest wall expansion:

Single breath count:

CVS:

RS:

P/A : Others:

## NERVOUS SYSTEM EXAMINATION

HMF :

Consciousness:

Speech :

MMSE:

CRANIAL NERVES EXAMINATION:

Fundus

Extra ocular movement

Nystagmus

Pupil

Facial weakness: U/L B/L

Palatal movement

Gag reflex

Dysphonia

Neck muscle weakness

SPINO MOTOR SYSTEM:

Bulk :

Tone: Right Upper Limb - Normal / decreased

Right Lower Limb - Normal / Decreased

Left Upper Limb - Normal / Decreased

Left Lower Limb - Normal / Decreased

| | Right | Left |
|---|---|---|
| Power: | | |
| Neck | | |
| Trunk | | |
| Shoulder Joint | | |
| Elbow Joint | | |
| Wrist Joint | | |
| Hand grip | | |
| Hip Joint | | |
| Knee Joint | | |
| Ankle Joint | | |
| Toe grip | | |

Plantar response :

Deep Tendon Reflexes

Biceps jerk

Triceps jerk

Supinatorjerk

Knee jerk

Ankle jerk:

SENSORY SYSTEM :

Pain /Touch/Temperature

Vibration sense/joint position sense

Rhomberg's sign

STANCE & GAIT :

CEREBELLAR SYSTEM :

AUTONOMIC SYSTEM:

PERIPHERAL NERVE EXAMINATION:

Signs of meningeal irritation

## INVESTIGATIONS

Hb:

TC: ----------, DC: N- L - M - E - B -

ESR ;

Peripheral smear :

Blood Sugar: Random ------- Fasting------- Postprandial-------

(Mgms%)

Urea:

Creatinine :

Sodium:

Potassium :

Total Cholesterol :

ECG

CSF analysis:

Biochemistry:

Glucose
Protein
Chloride
Globulin

Cell count:

HIV Screening:

**Nerve conduction study:**

MNC

| NERVE | LATENCY (ms) | AMPLITUDE (µV) | NCV (m/s) | F LATENCY (m/s) |
|---|---|---|---|---|
| Rt Median | | | | |
| Rt Ulnar | | | | |
| Lt Median | | | | |
| Lt Ulnar | | | | |
| Rt Peroneal | | | | |
| Rt Tibial | | | | |
| Lt Peroneal | | | | |
| Rt Tibial | | | | |
| Rt Orbicularis oculi | | | | |
| Rt Orbicularis oris | | | | |
| Lt Orbicularis oculi | | | | |
| Lt Orbicularis oris | | | | |

SNC

| NERVE | LATENCY ( ms) | AMPLITUDE ( µV) | NCV (m/s) |
|---|---|---|---|
| Rt Median | | | |
| Rt Ulnar | | | |
| Lt Median | | | |
| Lt Ulnar | | | |
| Rt Sural | | | |
| Lt Sural | | | |

Inference: Normal /Demyelinating/Axonal/Mixed Pattern

Other :

# KEY TO MASTER CHART

| | | |
|---|---|---|
| Asc. | - | Ascending paralysis |
| Des. | - | Descending paralysis |
| S | - | Symmetrical |
| D P | - | Distal paresthesia |
| D W | - | Distal Weakness |
| P W | - | Proximal Weakness |
| Q | - | Quadriparesis |
| LRTI | - | Lower Respiratory Tract infection |
| URI | - | Upper Respiratory infection |
| ↓↓ | - | Depressed Reflexes |
| Đ | - | Demyelinating type |
| PHT | - | Postural Hypotension |
| U RET | - | Urinary retention |
| M-F | - | Miller Fisher Variant |
| P-C-B | - | Pharyngo Cervico Brachial variant |

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